Epigenetics and the Disabled Self:

Finding an Ethical Narrative in the Shifting Boundaries of Molecular Science

Katie Saulnier, Biomedical Ethics Unit, Department of Philosophy, McGill University, Montreal

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For my sister, Hannah Saulnier, who keeps me chasing down the questions that can build a better world;

And for my partner, Rabbi Andrea Myers, who reminds me every day of the power of nurture.

Contents

Abstract

English

Epigenetics is the study of mechanisms, including environmental and social factors, that influence and modify gene expression. The field has engaged the imaginations of ethical, legal and social issue (ELSI) researchers, particularly because the epigenome, unlike one's genetic code, is potentially both heritable and reversible. However, one perspective from which this scholarship could benefit is strangely absent: that of critical disability theorists.

Disability theory enables us to interrogate our preconceptions of what it means to be "well", and of the standards by which a society demarcates and interacts with bodies that fall outside a given norm. In chapter one, I outline the ways in which scientific theories emerge from their social context and examine how epigenetics has inherited the pathologizing influence of twentieth century biomedicine. In chapter two, I introduce models of health and embodiment emerging from disability theory and consider how they can help shed light on how epigenetics discourse is not only emerging from but contributing to this pathologization of disability. Finally, in chapter three, I use the example of the history of autism research narratives to demonstrate how theory can crystalize into real harm, focusing particularly on the contribution of epigenetics to the attribution of "blame" to mothers for their offspring's health.

French

L'épigénétique est l'étude des mécanismes, incluant les facteurs environnementaux et sociaux, par lesquels l'expression des gènes est modifiée. Ce domaine de recherche suscite l'intérêt parmi les chercheurs étudiant les aspects éthiques, juridiques et sociaux de la santé humaine, en partie parce que l'épigénome, contrairement au code génétique, pourrait être non seulement héritable, mais réversible. Cependant, le domaine manque encore la perspective cruciale des chercheurs travaillant dans le champ d'étude de la théorie sur les personnes handicapées.

La théorie sur les personnes handicapées nous aide à interroger nos idées préconçues au sujet du bien-être et des standards par lesquels notre société définit et interagit avec les personnes qui ne se conforment pas aux normes. Au chapitre un, j'expose comment les théories scientifiques se développent en fonction de leurs contextes sociaux et j'analyse comment l'épigénétique a hérité de la tendance de la biomédecine du vingtième siècle de parler en termes de pathologie. Au chapitre deux, je présente les modèles de la santé et de l'intégrité corporelle résultant de la théorie sur les personnes handicapées et je considère comment ceci nous démontre que le discours épigénétique, non seulement émerge de l'histoire de la pathologisation en biomédecine, mais contribue aussi à celle-ci. Finalement, au chapitre trois, j'emploie l'exemple de l'histoire de la recherche au sujet de l'autisme pour démontrer comment les théories peuvent générer de vrais préjudices, en mettant l'emphase sur l'attribution de responsabilités aux mères pour la santé de leurs enfants.

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Introduction

In 2003, the Human Genome Project announced that they had completed a map of the human genome, ushering in the genomic era of human health. Thirteen years in the making, the project was heralded as the rise of molecular medicine, poised to uncover the roots of disease and to generate new, more personalisable approaches to health and disease. At the same time, as the numerous ethical, legal, and social implications (ELSI) of this feat became apparent, bioethicists worried about how this knowledge might eventually lead to discrimination, deterministic views of disease, and decreased attention to the social context surrounding health. In particular, researchers feared that understanding the genetic roots of disease might lead to a fatalistic view of personal responsibility for one's own body, and pull attention and funding from the social factors that influence health.

Enter modern epigenetics – the study of mechanisms that influence and modify gene expression – which emerged alongside genetics, albeit with less fanfare. Beginning under Conrad Waddington in 1942, epigenetics did not take on its modern definition until the 1990s, and a tentative consensus definition (which continues to evolve) was reached only in 2008. (Slack 2002). This definition – a "stably heritable phenotype resulting from changes in a chromosome without alterations in the DNA sequence" – has also engaged the imaginations of ELSI researchers, particularly because the epigenome, unlike one's genetic code, is potentially both heritable and reversible. Epigenetics is increasingly being employed to examine justice in health, intergenerational justice, responsibility for both individual and public health, and access to prevention and treatment.

The Literature

With every major shift in our understanding of human biology come new implications for the assignment of normative value to people and their bodies. These values are social and cultural;

moreover, they are not static. The genomics era shifted the focus on health and wellness to the molecular level, bringing with it the language of "good genes" and "bad genes" as shorthand for desirable and undesirable bodily traits, both in terms of health and in terms of adherence to socio-cultural aesthetic standards. Now, researchers are already hailing the start of a *post*-genomic era, one that rejects rigid conceptions of the body in favour of understanding the body as mutable and permeable, intrinsically tied to its environment (Meloni 2018). Such arguments, of course, predate genetics and epigenetics conversations, and will come as no surprise to social scientists and epidemiologists. However, the ability to quantify these relationships between body and environment at a molecular level provides new and persuasive forms of evidence of the entanglement of nature with nurture.

Some scholars are optimistic that this new evidence will lead to enhanced public health policies, joining with researchers in the field of Developmental Origins of Health and Disease (DOHaD) in order to argue for enhanced maternal and early life care in hopes of side-stepping potential epigenetic harms stemming from in utero and early life adversity. However, such policies, by their very nature, tend to contemplate and attempt to answer the question of who, ultimately, is responsible for human health at both the individual and social levels. Moreover, policymakers must define the types of bodies that are envisioned as the end-goal for these programs. The model advanced by DOHaD research is primarily one that focuses on epigenetic "deficits" – modifications that are seen to be the source of error, adverse affects, or disease risks in the human body (Richardson 2015). This, coupled with increased attention on in utero exposure as the temporal location of an individual's most drastic epigenetic modifications, means an increased scrutiny of parental choice.

From a scientific standpoint, it remains to be seen the degree to which our epigenome will

become a locus of optimization in health from the scientific standpoint. Many scholars are urging caution around the overenthusiasm accompanying epigenetics, pointing out the relative youth of the field and the fact that much of the evidence so far exists only in animal studies, which are particularly difficult to use as models for human *behaviours*. A shift in the language and tenor of health conversations, however, is already evident. In particular, epigenetics ELSI researchers have noted a rise in the responsibility for health attributed to mothers, with maternal bodies that are perceived to be providing an insufficient epigenetic environment being framed as "metabolic ghettos" (Wells 2012).

Epigenetics ELSI researchers have expressed concerns surrounding the responsibility that will be attributed to mothers, examining the problematic extrapolation from animal models to human maternal behaviours, the lack of attention paid to the contribution of fathers to their offspring's epigenomes, and an oversimplification of the social and cultural bases for certain behaviours and exposures (Hens 2017, Kenney and Muller 2017, Richardson 2015, Warin et al. 2015). However, underpinning this conversation, one that has drawn in researchers from gender studies, food justice, and biomedicine, is another, interconnected set of questions: In order to increase scrutiny on personal behaviours that create specific epigenetic modifications, we must first decide which epigenomes are "desirable". Much as a person may be said to have "good genes", who will decide what makes a "good epigenome"? And what impact will this have on those whose epigenomes are deemed "bad"?

The attribution of increased *value* to certain bodies over others through the language of health is not new. However, epigenetics adds a new dimension to this conversation by drawing together biological and social factors in concrete ways. In genetics, attempts to modify the genome via either gene editing or selective breeding are seen as dangerous at best and eugenics at worst.

The epigenome, in being perceived as less static and more mutable than the genome, is so far proving much more enticing (and more morally acceptable) as an area for human modification, even if the science is not yet there. It will be crucial for the discipline to take stock of what this will mean, particularly for those whose bodies have historically been perceived as carrying less value due to disability.

The Disability Gap

Surprisingly, a clear assessment of epigenetics ethical issues from the disability perspective has not yet emerged. A 2019 review found that epigenetics ELSI work is taking place in a wide variety of fields, from the expected areas of bioethics and science, technology and society (STS) to the fields of critical gender theory and radical geography (Dupras, Saulnier, and Joly 2019). However, the disability theory lens remains thus far absent from the field. In some ways, this may be a matter of priority. While certain theoretical aspects of epigenetics spark excitement for researchers looking down the road, many disability scholars, by necessity, focus their analysis on the now. Although the exact numbers are difficult to determine, the disability day of mourning project lists 139 deaths of disabled individuals by caretakers in 2018, mostly cases of homicide. Those with physical or cognitive disabilities face rates of sexual assault far higher than the rest of the population. This means two things: one, much disability scholarship focuses on combatting the harms that are already occurring; two, shifts in public perception of disability and "good" vs "bad" bodies via new science are not just matters of esoteric bioethics, but rather questions with real-world violent implications for already vulnerable individuals.

In allowing us to unpack the concept of disability and the socially generated normative values that are assigned to bodies, disability theory presents an important and overlooked area of analysis for understanding the assertion that epigenetics is fundamentally changing our

understanding of the relationship between nature and nurture, by showing how the definitions of health themselves always rely upon social constructs. Moreover, disability theory allows us to critique these social constructs and examine the ways in which the attribution of negative value to disabled bodies in turn creates further harm and vulnerability. Particularly at this moment, where we are seeing an explosion of new analyses within epigenetics ELSI literature that will shape the future directions not just of policy, but of the science that underpins that policy, it is crucial that we conduct a critical examination of the foundations of these assumptions. The absence of the disability lens over epigenetics discourse is therefore one that needs to be rectified.

Disability theory is already deeply immersed in questions surrounding the relationship between physical and social aspects of health, and has already examined many of the issues that come up repeatedly in epigenetics ELSI literature. Does epigenetics add an entirely new dimension to these questions? If so, where can the disability framework be most useful in providing answers? Rather than argue that epigenetics is bringing totally new ideas to the table, I will show how it is taking old problems of pathologization and moralizing around disabled bodies and bringing them along into a new field. I will argue that with epigenetics still in its infancy, insights from disability theory can be used to head some of these problems off at the pass, and to potentially shape epigenetics discourse in a way that incorporates these insights from the start, rather than having to apply them after the fact.

Chapter One

In chapter one, I will discuss what epigenetic discourse is contributing to changing conceptions of the value of disabled bodies. I will explain epigenetics, stigma and normative values in health, and argue that we place increased value judgments on wellbeing for things that we believe to be within an individual's control. I will show how the language of epigenetic harm and

epigenetic deficits both stem from certain historical normative claims about the body and contribute in turn to this normative discourse. I will also draw attention to the problem of presumed objectivity in scientific research, using the work of George Canguilhem on challenges to the alleged neutrality of medical sciences, as well as the work of Ludwik Fleck on proto-ideas to assess how much of the scientific presumptions made about epigenetics have emerged from a historical conflict between "nature" and "nurture" as foundations for human behavior and health. I will argue that conventions and power dynamics within the medical sciences shape the direction of emerging scientific fields, and show how this is being enacted in epigenetics. Finally, I will argue that in overvaluing molecular evidence for interactions between bodies and their environments while undervaluing existing research on these interactions from the social sciences perspective, we oversimplify the immense complexity of human health as a qualifiable experience, rather than simply a quantifiable state.

Chapter Two

In chapter two, I will elaborate on the connection between epigenetics discourse and preconceived notions of embodiment and the value of bodies, and propose critical disability theory as a way of dismantling the presumptions at work in current epigenetic discourse and of building alternative frameworks. I will present a variety of models taken from disability studies and critical disability theory, and will examine how emerging themes in epigenetics might be analyzed through and within these models. I will engage particularly with disability literature that examines normative judgements about bodies and the work of developing approaches to human health that do not rely on the pathologization of bodies that lie outside statistical norms. In particular, I will use the disability lens to deconstruct the conflict between acknowledging the need for access to nurturing environments (both social and physical) and the need to avoid demonizing bodies that

represent the outcome of environmental adversity. Finally, I will use a disability critique to interrogate the trend toward biological neoreductionism in epigenetics, arguing instead that epigenetics should push us to consider the value of body *as experienced*.

Chapter Three

Chapter three of this text will function as proof of concept for Parts I and II and will provide concrete cautionary examples of the impact of pathologization on disabled individuals. Starting from a framework that presupposes the value of a neurodiverse population, I will present a narrative analysis of the autistic experience of harm from the normative values that have accompanied the "deficit" model of cognitive well-being, focusing on how these narratives are acted out upon the individual. I will begin with the history of shame and blame involved in both autism research and media reporting on autism, beginning with the causal assumptions made by early autism researchers. I will show how initial conceptions of maternal responsibility for autism (via "refrigerator mother" rhetoric) set the stage for more modern discourse of shame and responsibility, from the 2007 "Ransom Notes Campaign" of the New York University Child Study Center to current conflicts between autistic advocates and Autism Speaks. I will show that the shift from autism as environmental to autism as genetic (the MSSNG project), culminating in autism as epigenetic, via maternal exposures, risks losing ground won by autism advocates by return to prevention and curative rhetoric (Volkmar 2019). Finally, I will show the impact of this discourse on autistic individuals and argue that incautious epigenetic autism research risks exacerbating existing harms to autistic individuals and their families, including high rates of depression, anxiety, and suicide, as well as caregiver abuse spanning from potentially fatal curative attempts and infanticide.

Methodology

The beginning literature review for this thesis was undertaken partly under the auspices of my work at the Centre of Genomics and Policy reviewing the current state of epigenetics ELSI research with Dr. Charles Dupras and Prof. Yann Joly, as part of our work for the bioethics subcommittee for the International Human Epigenome Consortium. Chapter one draws extensively from this review, which was later developed into a paper published as "Epigenetics, ethics, law and society: A multidisciplinary review of descriptive, instrumental, dialectical and reflexive analyses" in Social Studies of Science (Dupras, Saulnier, and Joly 2019). The literature search for this work began in 2016, using keywords such as 'epigenetics', 'DNA methylation', 'histone', 'ethics', 'law' and 'society'. It included articles published on the topic of epigenetics and ethical, legal and social scholarship in academic journals between 2000 and 2017. In particular for this thesis, I draw from the literature that touches on themes of personal and maternal responsibility for the epigenome, the challenges of bioreductionism in emerging epigenetics literature, and the potential for epigenetics to shape public policy. I then reviewed the literature that had emerged in 2018 and 2019, as well as literature examining the development of scientific ideas, focusing on the work of George Canguilhem, Ludvik Fleck, and Michel Foucault in particular.

In chapters two and three I draw largely from literature from critical disability studies and the neurodiversity movement. The work of Jackie Leach Scully on the intersection of disability theory and genetics was of particular help here. Throughout all three chapters I engage in a narrative approach, looking to examine the ways in which we communicate and tell stories about bodies and health. Drawing from critical disability theory, I employ a framework that presupposes the value of disabled people, and that not only seeks to observe the impact of harmful narratives

on individuals and communities, but that is committed to praxis. This thesis is intended to move the reader not only to thought, but to action, if only through encouraging them to push back in their own work on the idea that scientific concepts exist in a vacuum.

Research findings

As I will argue in the pages to come, epigenetics as an emerging field is already showing a tendency to feed into harmful narratives around the value of certain bodies over others. Disability theory is exceptionally well-suited to helping us understand why this is a problem, by highlighting how we assign normative values to bodies in a way that is actively harmful, and how to stop it. In particular, epigenetics scholars who are concerned with the employment of "blame" rhetoric in their field can benefit deeply from understanding how the attachment of moral value to the development of illness is intrinsically tied to the attribution of negative value to disabled bodies. Moreover, the intense focus on epigenetics researchers on their field as an answer to the question of nature versus nurture is deepening this concern. However, thus far disability theorists have refrained from engaging in depth with epigenetics literature, and epigenetics ELSI scholars have by and large ignored disability theory insights. In focusing on the environment at a molecular level, epigenetics runs the risk of entrenching value-judgements that stem from power imbalances in medical fields. Meanwhile, prevention of these harms means interrogating assumptions about the social factors that underpin our conflation of health and "goodness". Major shifts to these values after the fact take significant energy; it is better to shift focus now than after the fact, when the narrative has become entrenched. With serious epigenetics research still in its infancy, now is the perfect time to begin re-writing this story.

Chapter 1: Epigenetics and the Myth of Scientific Neutrality

"When we think of the object of a science we think of a stable object identical to itself. In this respect, matter and motion, governed by inertia, fulfill every requirement. But life? Isn't it evolution, variation of forms, invention of behaviors? Isn't its structure historical as well as histological?" (Canguilhem 1989, 203)

What is Epigenetics?

Although research at the intersection of genomics and developmental biology began broadly under this name as early as 1942 with the work of Conrad Waddington, epigenetics in its current form appears in the 1990s, with a consensus definition on the phrase "epigenetic trait" being reached in 2008. This definition – a "stably heritable phenotype resulting from changes in a chromosome without alterations in the DNA sequence" – is of great interest to ELSI researchers;, the epigenome, unlike one's genetic code, is potentially both heritable and reversible (Berger et al. 2009, Tarakhovsky 2010). Among its branches are environmental and social epigenetics, examining respectively how environmental factors and early life experiences cause epigenetic alterations. Epigenetics is becoming increasingly relevant to conceptions of justice in health, engaging questions of intergenerational justice, responsibility for both individual and public health, and access to prevention and treatment. In particular, bioethicists have expressed interest in epigenetics as providing evidence on a molecular level for the deep interrelatedness between health outcomes and family and community environments. Epigenetics shows us how environmental and social context affects the individual at their core, and suggests a much higher level of biosocial porosity than was imagined in the genomic era (Meloni 2015).

The story of epigenetics as told by Conrad Waddington in 1942 was not just a story of an emerging science, but rather one that considered the deep links between the human body and its socio-cultural environment. Waddington's work, which led to his characterization as "the last Renaissance biologist", spanned palaeontology, population genetics, developmental genetics,

biochemical embryology and theoretical biology (Slack 2002, 42). Interestingly, modern approaches to epigenetics exhibit a much starker divide between those who see it as changing the way we imagine the body, and those who see it as providing evidence for the importance of information gleaned from the social sciences. Indeed, despite being touted as helping to finally bridge the nature-nurture divide, bioethicists have expressed a concern that epigenetics will in fact serve to further entrench a molecularized understanding of the human body (Dupras, Saulnier, and Joly 2019).

No *Really*, What is Epigenetics?

The jury is still out on whether epigenetics functions as a field overseeing a new era of science; a massive overhaul of genetics; or simply a more nuanced way of understanding how genetics and the environment interact. Early researchers in the field debated the value of epigenetics as a gene-centric science versus one that examines dynamic systems, asking the fundamental question "Does epigenetics today lead to such a paradigmatic shift in biology?" (Van de Vijver, Van Speybroeck, and De Waele 2002, 2). Seventeen years later, this conversation has not reached any obvious resolution. A 2018 paper entitled "A user's guide to the ambiguous word 'epigenetics'" noted that the continued lack of clarity on the meaning and significance of epigenetics has led not only to an over-interpretation of its impact, but also a "domestication" of the term by those wishing to use a certain conception of genetic non-determinism to develop "commercial offerings such as epigenetic yoga, epigenetic face cream and epigenetic anti-dandruff shampoo" (Greally 2018).

The question then, is not merely how or if epigenetics differs from genetics in terms of its biological implications, but also with regard to its implications for knowledge translation and,

more broadly, its impact on our understanding of human health, and what it means to be a body existing in a particular environment. As Van de Vijver et al. note:

> molecular biology is not relieved from questioning its epistemological and metaphysical interests and purposes that determine its ways of interacting with living systems, it is not freed from defining its "knowledge attitude" towards living beings. (Van de Vijver, Van Speybroeck, and De Waele 2002, 5)

These concerns are increasingly being taken on by ethical, legal, and social issues (ELSI) scholars. In "Serving Epigenetics Before Its Time", Juengst et al. argue that society's rush for rapid translation of basic biological science into new approaches to health is evident in epigenetics research, leading to premature claims of health breakthroughs and unnecessary pressure on future parents (Juengst et al. 2014). In a response to this article, Joly et al. expand on Juengst's concerns to note that researchers bear a responsibility to minimize this "culture of premature risk messaging" (Joly et al. 2016).

Although these are crucial questions for the field to continue to engage with as it evolves, a conclusive answer on the relationship between epigenetics and other fields of human health is far beyond the purview of this chapter. Moreover, I will not put forth an argument here regarding whether or not the study of epigenetics lives up to its hype (even as I write this, the discourse is shifting; the microbiome is now the subject of similar debate.) Instead, accepting this ambiguity as part and parcel of working in an emerging field, I will look at the historical, social, and cultural ideas that underpin this debate around the significance and future directions of epigenetics. Focusing particularly on the work of Georges Canguilhem, Ludwik Fleck, and Michel Foucault in the first half, I will consider the ways that scientific fields are shaped by the social context and power dynamics from which they emerge. Then, in the second half, I will elaborate on how epigenetics' biological and social predecessors are shaping the current discourse, paying attention to what is present and what is missing in today's ELSI scholarship.

The Persistent Myth of Scientific Neutrality

The Historical and the Histological

The practice of medicine relies on the establishment of thresholds and norms. As Georges Canguilhem notes in the first line of *The Normal and the Pathological*, "[t]o act, it is necessary at least to localize"; without some definition of *health*, it would be impossible to practice health*care* (Canguilhem 1989, 39). Canguilhem wrote the first edition of *Essai sur quelques problèmes concernant le normal et le pathologique* in 1943, which was re-published as *Le normal et le pathologique, augmenté de Nouvelles réflexions concernant le normal et le pathologique* in 1966 and translated into English as *The Normal and the Pathological* in 1989. As such, his first edition came only a year after Waddington's first attempt to publicly define the epigenome in recognition of the interplay between environment and genetic expression. Nevertheless, it is just such a conception of a body's "terrain" – "the state of an organism with regard to its resistance to pathogenic agents or its predisposition to different diseases" that Canguilhem argues complicates any simplification of health sciences (Canguilhem 1989, 305). To merely evaluate the body *qua* body is to miss out on the infinite complexity of human experience, and is, on an individual level, more useful as a thought experiment than an approach to health; to evaluate the body *in situ* is to risk conflating statistical observations and moral judgements.

To make note of a difference does not mean that this difference will inevitably come to be pathologized; however, in the medical context, such pathologization becomes significantly more likely, for reasons I will examine below. To pathologize something is not only to make note of difference, but to say that this difference is something that needs to be fixed. This is why, for instance, the removal of homosexuality from the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders held such weight (and why transgender communities

eagerly await the removal of the diagnosis of gender dysphoria). "Pathological", Canguilhem remarks, "implies *pathos*, the direct and concrete feeling of suffering and impotence, the feeling of life gone wrong" (Canguilhem 1989, 137). Canguilhem saw this as a shift toward pathologization as a sign of medicine moving from the idea of balance between health and sickness used by the Greeks, to an idea that we can eventually learn enough for disease to vanish altogether. Pathology, then is viewed as a natural extension of the emergence of physiology as a discipline (Canguilhem 1989, 42).

The conviction behind physiology and its accompanying pathology is that there not only exists a norm, but that this norm can be scientifically restored (Canguilhem 1989, 42). While it is true that the normal and the pathological, in terms of modern medical practice, must be in some way defined in the pursuit of remedying the pathological, it is inaccurate to claim this as a mere neutral scientific advancement of knowledge:

> In the course of the nineteenth century, the real identity of normal and pathological vital phenomena, apparently so different, and given opposing values by human experience, became a kind of scientifically guaranteed dogma (Canguilhem 1989, 43)

This is not, either, intended to place the criticism for pathologization entirely at the feet of health sciences. The philosophical value of the ideal type, after all, traces back to the ancient Greeks, and the idea of some sort of neutrality in scientific methodologies has infiltrated everything from pedagogy to moral philosophy.

While interdisciplinary studies have, in recent years, grown in respectability, so has the contrast between the STEM (Science, Technology, Engineering, and Mathematics) fields and the study of social sciences, arts, and humanities. Waddington's type of broad interdisciplinary study, or Canguilhem's history of the histological, become rarer as the sheer volume of available knowledge drives specialization deeper. Epigenetics scientists have accused bioethicists of

overstating potential concerns about their work, while bioethicists worry that scientists ignore the social framing of their own work. Both are correct. As an additional challenge, scientists and social scientists have long conflicted over the types of methodologies and evidence viewed as necessary and sufficient for something to be deemed relevant to the field. Some of both the hype and discomfort around this hype that exists around epigenetics stems from the assertion that epigenetics allows us to prove at a molecular and more refined level what epidemiologists and social scientists have already claimed with regard to adversity and health. Sitting central to both molecular sciences and social sciences, the question of what degree of evidence will be sufficient to declare a link between environment and health is one that plagues both scientists and social scientists. This question – and its lack of obvious answer – have contributed to the long-standing debate between the value of nature (mostly viewed in modern terms as genetics) versus nurture (including both environmental exposures and social context) in determining health outcomes.

Epigenetic Ancestry: The Nature-Nurture Debate

The question of whose evidence will be privileged in this debate is, in part, a methodological one (Tabery 2009). Statistics and norms form an important and necessary part of medical science, and of setting benchmarks for human health. However, the assignment of value to these norms requires a human assessment. The identification of an epigenetic biomarker is one thing; the decision to attempt to reverse it is another, and one that requires that we assign value to particular epigenomes. This type of value assessment depends heavily on what we consider to be an ideal body, or a body in good health. In epigenetics, the emerging discourse around this tends to focus not just on finding ways to measure or analyze interactions between the body and its environment, but more broadly to make claims that epigenetics is poised to finally put to rest centuries-old questions about these interactions.

This is not a point of criticism regarding epigenetic researchers, per se. As the work of early 20th century physician and biologist Ludwik Fleck explains, scientific ideas are shaped by what Fleck terms "proto-ideas". Using a form of comparative epistemology, Fleck argues, inescapably, that "What is already known influences the particular method of cognition" (Fleck 1979, 38):

> Whether we like it or not, we can never sever our links with the past, complete with all its errors. It survives in accepted concepts, in the presentation of problems, in the syllabus of formal education, in every life, as well as in language and institutions. Concepts are not spontaneously created but are determined by their 'ancestors' (Fleck 1979, 20).

There is no more obvious ancestor for epigenetics than the grand nature-nurture debate (Tammen, Friso, and Choi 2013, Crews et al. 2014, Simmons 2011, Powledge 2011, Music 2017). Indeed, epigenetics in popular culture and scientific reporting has frequently been hailed as the answer to the longstanding argument over which one should be given more consideration. As one writer of popular science remarks in the provocatively titled "The Epigenetics Revolution", "we are finally starting to unravel the missing link between nature and nurture; how our environment talks to us and alters us, sometimes forever" (Carey 2012, 7).

Underneath the epigenetics hype, then, is the presumption not just that epigenetics holds the key to the nature-nurture debate, but that this debate in and of itself is the correct question. The nurture aspect of the question itself can be traced in philosophy to Locke's *An Essay Concerning Human Understanding* (1690) as the foundation of the "blank slate" view, which describes the mind at birth as being blank, later to be filled in by experience (Cosmides and Tooby 2013, Locke 1690). The nature side of the debate, by contrast, is commonly attributed to the excitement surrounding Darwin's publication of *On the Origin of Species* (Cravens 1978, Darwin and Darwin 1859). Over the twentieth century, finding the "correct" midpoint between these two ideas – we start from nothing and experience fills in the gaps, or our lives are predetermined – has drawn in

academics from behavioural psychology, theology, philosophy, and beyond. What the underlying proto-idea of nature versus nurture seems to have contributed to the epigenetics debate is largely to presume that this is the most important question that epigenetics can answer for us. Moreover, as epigenetics research is firmly rooted in the area of molecular sciences, it tends toward privileging methodologies and outcomes from science. As a result rather than illuminating the links between nature and nurture, epigenetics has proven instead to be prone to subsuming nature *under* the category of nurture, with the bioreductionist argument that in the end, it all comes down to molecular science after all.

Power Dynamics in Health Sciences

Of course, it is not just a matter of unpacking *which* historical ideas underpin the direction of emerging scientific fields like epigenetics, but also *why*. Conventions and power dynamics within the medical sciences shape the directions of these debates. As I write above, epigenetics thus far seems to lean toward a preference for privileging the more traditional scientific side of this equation, the result of which may be a particular turn toward pathologization. Drawing from Foucault, Henk A. M. J. ten Have argues that geneticization has itself taken on the role of a heuristic tool in moral debate. As ten Have notes, Foucault demonstrated that we live in a culture that stresses normalization, as well as a focus on reducing the body and accompanying biomedical claims to a Cartesian reductionism – something that I argue is problematized by epigenetics' focus on drawing together the genome and its environment (Foucault, 1970, 1973). Medical practice creates false dichotomies between illness and health; under ten Have's reading of Foucault, this is not merely a classification within the field of medicine, but a method of creating and maintaining social order (ten Have 2001). The nineteenth century shift toward geneticization is then mirrored by an epistemological shift that privileges an understanding of the interior of the body. Unlike an

approach to medicine that focuses on symptomology, this inward shift is knowable only by those with specific expertise, resulting in a shift in the value of (genetic) medical knowledge as an otherwise unknowable specialization. The locus of power rests, not so much with individual doctors, but with medical culture itself: "Medical knowledge transformed in modern societies into biopolitics, whereas at the same time biopolitics transformed human beings into subjects of scientific control" (ten Have 2001). The practice of mapping the body genetically, in "[p]resenting the body as a machine comprising interchangeable parts […] take[s] apart processes, not only distinguishing the psyche and soma but dismembering 'physical health' itself into separate territories to be divided and conquered" (ten Have 2001). In doing so, the body is even further objectified and the concepts of health or illness are separated from the person, casting Foucault's medical gaze on the genome itself, and centering the power of knowing the body with the clinical geneticist (Lippman 1992, 1470).

Lies, Damn Lies, and Statistics

One blatant example of human normative presumptions and the establishment of scientific standards that come to be taken as objective truth is the evolution of the body mass index over the latter half of the 20th century. In 2004, computational errors led to a report that more than 400,000 American deaths annually could be attributed to obesity, trailing only smoking as the leading cause of preventable death (Mokdad et al. 2004). This information was reported by the Centers for Disease Control and Prevention (CDC) in the *Journal of the American Medical Association* (JAMA). A later correction of these errors reduced the estimate *fifteen-fold* (Flegal et al. 2005). However, this correction was not widely publicized. As Fleck reminds us, "many very solidly established scientific facts are undeniably linked, in their development, to prescientific, somewhat hazy, related proto-ideas or pre-ideas, even though such links cannot be substantiated" (Fleck

1979, 23). The corrections to these statistics were published, but the ideas had already taken hold, in part because they confirmed the existing sense that to be obese *must* be to be not only unhealthy, but immorally so; a correction of the science was not enough to undo this idea.

As Linda Bacon reports in *Health at Every Size*, the idea that obesity kills has since been the "backbone of the federal health campaign" (Bacon 2010, 125). Every fat individual is familiar with the result; while bullying and discrimination for weight-based causes remains high, another tool has been added to that arsenal: that of "concern-trolling": "I'm just worried about your health." However, a number of studies, including an epidemiological investigation with a cohort of 1.7 million Norwegians, have shown highest life expectancies to be in those that are overweight by current medical standards, and the lowest to be among those who are underweight by those same classifications (Waaler 1984). Instead, a correlation is shown between the effects of perceiving oneself as and being perceived as being overweight; "[i]n other words, *feeling* fat has stronger health effects than *being* fat" (Bacon 2010, 131). So why do conflations of weight and poor health persist, and why are we seeing epigenetics researchers carrying over old incorrect data into a brand new field?

The answer lies partly in understanding that obesity is considered not only medically but morally problematic, making any discussion of connections that do exist between weight, environmental influences on weight, and health outcomes extremely difficult to undertake in a value-neutral way. As a result, epigenetic modifications associated with obesity shift from mere molecular differences to molecular deficits, which in turn feeds back into pathologization of the condition itself. Causal links between the epigenome and obesity are a hotly researched topic. While the fact that an individual's weight and metabolism are determined by a combination of environment and genetics is extremely well-recognized in the literature by this point, the added

moralization permitted encouraged in epigenetics writing – the "personal responsibility" rhetoric – adds to this dilemma (Herrera, Keildson, and Lindgren 2011). For instance, one epigenetics researcher begins a paper with the line "[o]besity is among the leading causes of death in the US and Western Europe, with over 110,000 and over 270,000 deaths per year in US and EU countries, respectively, attributed to excess adiposity" before going on to list the actual causes of death as cardiovascular diseases, type II diabetes, cancer, renal failure, chronic pulmonary diseases (known to be correlated with, but not necessarily caused by, certain kinds of weight gain). He then ends his paper by arguing that rising obesity rates constitute a "national security threat" for their impact on a country's military recruitment potential (Niculescu 2011).

Fatness has thus been pathologized within medical sciences; rather than being a piece of data that can contribute to our full understanding of a person's health, it has come to be considered in and of itself not only a health problem, but a moral one (Guthman 2013). Of course, to examine fatness *only* as a social construct is to ignore the lived reality of the body not only at the molecular level as examined in epigenetics, but as a real biological entity that straddles the conceptual and material experiences. Megan Warin argues that "in developing arguments that only critique representations of obesity or fat bodies, social science scholars have maintained and reproduced a central dichotomy of Cartesian thinking – that between social construction and biology" (Warin 2015). Epigenetics has much to contribute to our understanding of human weight and health. The challenge occurs when we recognize the number of presumptions that underlie both social sciences and scientific claims about weight. Normativity becomes a barrier to a complete and accurate scientific exploration of the topic, whereby pathologization leads not only to social harms, but to bad science.

This kind of normative circularity has long been recognised within queer theory (with which critical fat studies and critical disability theory share common roots) (Cuonzo 2013). Cuonzo describes descriptive circularity as the phenomenon that occurs when "evidence is amassed in a way that already assumes a conclusion that will be eventually drawn from the data" (Cuonzo 2013, 223). Data that falls outside of this normative circle is dismissed as an outlier. Recall the central issue of Bacon's work, and consider how this is currently playing out in literature on the epigenetics of obesity. As Megan Warin notes, "Social scientists, particularly those trained in social constructionism, will come to know obesity as the product of medicalization discourses, and be critical of the pathologization and blaming of fat bodies" (Warin et al. 2015, 54). The outcome of this is a standoff between scientists and critical theorists, where neither is willing to cede ground: scientists for the intrusion of moral and social discourse into what is perceived to be a question of unbiased measurement, critical theorists for fear that acknowledging the places where the science is of use will simply serve to deeper entrench the pathologization discourse.

The Socio-Cultural Building Blocks of Epigenetics and Their Implications

Epigenetics, Pathologization and Normative Assumptions

Despite its newness as a field, we are already seeing these themes of pathologization and normative assumptions arising in epigenetics. For instance, epigenetics scholars are already centering research outputs on the idea of epigenetic "deficits" or "defects". As we will examine more closely in the next chapter, the potential for stigmatization and discrimination in epigenetics is closely tied to concerns about embodiment. Although emerging research offers us new insights into epigenetic impacts of discrimination, at the same time it risks a reductionist view of the biology-social interaction that could further entrench existing stereotypes and worsen discrimination against marginalized populations. In some areas of research, this same caution

raised by ELSI scholars is already being reflected in scientific research, such as in the case of geneticists who hesitate to explore the transgenerational implications of epigenetics for fear of repeating historical discrimination that emerged out of badly executed genetic research in the same area (Graham 2016).

In particular, new epigenetic understandings of racial health disparities have led researchers to express apprehension about the challenges inherent in undertaking analyses of race in health without reintroducing harmful rhetoric about the concept of biological race (Saulnier and Dupras 2017, Pickersgill et al. 2013). At the same time, however, authors are noting that epigenetic research may help to better understand the biologically inherited effects of racism (Sullivan 2013). For instance, an epigenetic model of racial health disparities has been suggested with regard to the marked prevalence of premature birth and cardiovascular illness among African-Americans. Particularly given the practice of normalizing privileged bodies (e.g. measuring obesity based on norms seen in white bodies), researchers therefore recommend caution in viewing biological difference as a cause of disparate health outcomes, framing these disparities instead as the embodied result of discrimination (Guthman 2013, Kuzawa and Sweet 2009). A balance is therefore sought between the recognition of the problems generated by past racialized research, and the potential for a new "plastic and biosocial" view of race that considers both the social and biological evidence (Meloni 2017, Dupras, Saulnier, and Joly 2019).

One concern surrounding epigenetics and the risk of discrimination lies in the potential for new modes of and reinforced ideas about personal responsibility in for the body. While epigenetic evidence of the Developmental Origins of Health and Disease (DOHad) are seen as promising (as discussed above) in the area of preventive policies, at the same time these policies disproportionately impact already vulnerable groups by normalizing particular (privileged)

environments, while attributing epigenetic "contamination" to "abnormal" practices (e.g. the denigration of cultural practices that result in epigenetic changes) (Mansfield and Guthman 2015, Guthman 2013). The very potential for environmental epigenetics to allow us to measure biomarkers of social inequality in new ways would lead some authors to worry about the development of new measurements of "normality", leading to preventive policies that target the health of vulnerable populations in a form of "epi-eugenics" (Juengst et al. 2014). In addition the implications for racialized populations, women and sexual minorities as well as socio-economically underprivileged individuals, face the possibility that our growing understanding of the bio-social interactions will lead to more intense scrutiny and increased blame for behaviours that deviate from the new norm (Katz 2018, Lock 2013, Niewohner 2015).

Epigenetics, Stigma, and Responsibility

Discussion of the ethical, legal and social implications (ELSI) of epigenetics has evolved and grown significantly over the last several years. In a comprehensive multidisciplinary review of emerging themes and analyses in epigenetics ELSI discussions, Dupras et al. named nine emerging themes across the literature. Of these, nearly half touched broadly on a particular question: as we learn more about the science behind epigenetics, who will come to be held responsible for either generating positive epigenetic effects, or preventing the development of those epigenetic markers perceived to be harmful? (Dupras, Saulnier, and Joly 2019). Scholars investigating the meaning of epigenetics for public health demonstrated an interest in how this might be harnessed for greater social responsibility, while scholars examining epigenetics' potential influence on reproduction and parenting discourse showed more trepidation on the topic of responsibility. Researchers looking at epigenetics and political theory as well as epigenetics, stigmatization, and discrimination similarly exhibited concerns around how personal and state

responsibility for health will be reallocated in the face of epigenetic evidence that environmental factors both under and outside of the individual's epigenetic control may contribute significantly to health outcomes. In particular, the perception that epigenetic modifications, in altering genetic expression, not only influence health and disease but also may make fundamentally identity-forming changes to the individual, adds a heightened sense of urgency to the question of who should be held responsible for those changes.

Among those papers included in the 2019 review, there were both optimistic and cautionary stances on emerging responsibility discourse. First, there are those who argue that in allowing us to see how social and environmental inputs affect the body on a molecular level, we will be able to provide better evidence for public health initiatives (Rial-Sebbag et al. 2016, McBride and Koehly 2017, Drake and Liu 2010). This argument is particularly prevalent amongst DOHaD scholars, who investigate the preconception, fetal, and early life origins of given health outcomes. It is important here to note that knowledge of a connection between environment, particularly early life environment, and health outcomes, long predates epigenetics. Social scientists in particular have noted the ways in which social programs that target poverty or childhood nutrition, programs that work to improve access to prenatal care, and other approaches that generally target the provision of an improved level of early life care all have lifelong consequences for both physical and mental health (M'hamdi et al. 2018). The hope is that as we become more capable of pinpointing specific epigenetic variants in order to understand how, for instance, a variant that is associated with development of a certain type of cancer is also associated with the lack of a specific nutrient as a child, we will be better placed to advocate for the implementation of early intervention programs. Moreover, these programs will be able to work more effectively, as they will be able to target those factors where we have tangible evidence of health implications (Hedlund 2012).

This first perspective largely envisions a state or community-based concept of responsibility, where epigenetic harms can be reduced at a population-wide level. It is perhaps not surprising then that this potential is often highlighted by researchers who draw on epidemiology, and who see epigenetics' potential in arguing for a communal responsibility for better epigenetic environments. The second perspective that is raised in the literature is one that focuses instead on personal responsibility. In particular, bioethicists warn that if we are not careful in how we communicate around epigenetics and preconception, fetal, and infant health, much of this responsibility will, in practice, get shifted at an individual level toward mothers (Kenney and Muller 2017). This worry reflects both historical and practical concerns. Certainly, mothers have often borne the brunt of blame for their children's health outcomes, often in ways that are disproportionate to (or even in contradiction with) medical evidence (consider, for example, researchers who long blamed the development of autistic children on emotionally withholding, or "refrigerator" mothers) (Richardson et al. 2014). More pragmatically, the mother is likely not only the person providing a uterine environment for the child, but also remains the parent most likely to be responsible for much of the early childhood care and decision-making. As a result, even as public policy researchers tout the potential contributions of epigenetics to population health, other bioethicists warn that epigenetics' increasing focus on mothers as loci of epigenetic responsibility may result in an increase in pressuring and shaming surrounding maternal decision-making (Warin et al. 2016).

At the intersection of the public health and maternal responsibility discourse lies the communication of health information to mothers. Numerous scholars have already examined the ways that even well-intentioned public health discourse and health communication in the context of maternal responsibility can cause harm, including the stigmatization of disability, stigmatization

of mothers, and a reluctance for mothers to seek help in changing potentially harmful behaviours (Zizzo and Racine 2017). Epigenetics is subject to these same concerns and also, because of the questions of heritability and epigenetic modifications as not only health-related but identity-forming, creates new maternal pressures. Early enthusiasm for the potential health implications of epigenetics has already concerned ELSI scholars, who question whether premature claims about the power of epigenetics in media reporting may do more to create alarm than improve health, and whether being bombarded with such information may make it difficult for individuals to feel confident in their decision-making abilities (Juengst et al. 2014).

Maternal Exposures

One of the primary concerns raised around epigenetics research is that it may end up heightening the surveillance of pregnant individuals or placing more restrictions on them through a sort of "molecular policing of behaviour" (Richardson 2015). This is of particular concern with the emergence of the field of study referred to as "maternal-fetal epigenetic programming", where the maternal body is framed explicitly as an adaptive environment for the fetus. Again, this conception of the body predates an understanding of the epigenetic mechanisms by which a fetus is altered through its environment. For instance, mothers who consume alcohol or tobacco while pregnant face significant disapprobation. This stigmatization has been shown to be linked not always or only to the level of health risk to the fetus, but rather to the formation of public moral judgements about "mothers behaving badly", leading mothers to feel reluctant to admit to usage or even to seek prenatal care that might expose substance use (Littler 2019, Grant et al. 2018, Millum et al. 2019). However, it is the way in which this discussion is evolving – or rather, the way in which it is carrying harmful stereotypes into a new and powerful way of scientific thinking – that has raised alarm. One of the topics that arises frequently in conversations around

reproductive autonomy is the challenge of balancing the reality that an individual's behaviours and environment while pregnant carry health implications for the fetus, while continuing to recognize the pregnant individual as a person unto themselves with a full range of bodily autonomy.

Language choice carries significant weight here. In the field of maternal-fetal epigenetic programming, the mother-as-environment has tended toward language that positions the maternal body primarily as a locus for epigenetic alterations, using language like "maternal capital" and, more alarmingly, "low-capital maternal environments" to refer not just to the environment in which the pregnant individual finds themselves, but to the actual maternal body itself (Wells 2012, 230). While the connection between adversity and reproductive health concerns is well-known, and has certainly already led to stigmatization of mothers, epigenetics further entrenches this stigmatization by focusing on the mother's body directly as the locus of fetal injury. Wells argues that mothers whose environments while pregnant are disadvantageous to the health of the fetus are "metabolic ghettos", as their bodies generate higher risks of perceived epigenetic harm (Wells 2012). This language of "ghettoization" brings to mind myriad pejorative socio-economic and racialized stereotypes, and hearkens back to the pre-epigenetics nature vs nurture debate in demonizing particular environments while failing to address the underlying causes of these disparities, and simultaneously framing the maternal body as a "mere" molecular environment for the fetus. Wells' justification of this terminology draws heavily on medieval European history and the Jewish Diaspora, as a way of indicating "social or spatial isolation" as social inequality extended to physiology (Wells 2012). However, while modern health disparities between African-American and white populations are later examined in the context of his metaphor, nowhere does he note the derogatory implications of the term's modern usage in generating and reinforcing harmful racial and socio-economic stereotypes when targeted toward black individuals and

communities (Richardson and Donley 2018). In drawing together the concepts of "maternal capital" and "metabolic ghettos", the maternal body in adversity is framed in and of itself as a second-class environment in which to foster a child. In using this kind of framing, epigeneticists risk generating shame and pressure to prioritize the optimization of one's body to produce better "maternal capital" over other desires or needs.

This risk is heightened precisely *because* this epigenetic framing is not new, but is instead contributing to entrenched historical visions of maternal bodies and emerging from the historical discourse surrounding motherhood, including preconceived ideas about the nature of perceived-as-binary sexes and their roles in reproduction and parenting. These ideas develop along a predictable path of unacknowledged overlap between the social and the scientific; one way to examine this is to watch the framing that is employed in epigenetic animal studies. In "Of Rats and Women: Narratives of Motherhood in Environmental Epigenetics", the authors examine how value-laden terminology and concepts around "maternal care" are translating into laboratory environments (Kenney and Muller 2017). Of crucial note is that a large portion of our current epigenetic understanding is confined to animal studies, in part because the field is so new; when scientists wish to examine epigenetic changes over a lifetime, this is much easier to investigate in rodents. One such famous study involved an examination of the epigenetic advantages that seem to be conferred upon rats who were licked and groomed more as pups. In the public reporting on this study, the researchers refer to such grooming as explicitly constituting *maternal* care, expressed as follows:

> So there are rats that do a lot of licking and grooming, and there are rats that do very little, but most rats are in between. So that resembles human behaviour as well. Right, you have mothers that are highly mothering and mothers that could not care less (Szyf 2008).

In framing the research in this way, there is a shift from the molecular understanding that rats who receive more grooming appear to express an increase in glucocorticoide receptor gene activity, to a very human social claim about the impacts of a mother's nurturing in the mental health outcomes of children. In doing so, notions of maternal care are simplified and decontextualized in potentially harmful ways, linking a health risk to a single molecule instead of complex social and environmental phenomena. This focus is particularly concerning in the context of extrapolating from animal studies to human implications, as it distracts from the myriad social factors that precede the maternal environment, while simultaneously endorsing particular stereotypes about maternal human behaviour, hearkening again back to the "refrigerator mother" rhetoric of the 1950s (Richardson 2015).

Thus far, I have discussed the intersection between maternal bodies and epigenetics. However, while the uterine environment is where we believe many important epigenetic changes take place, both the biological contributions of the sperm to the development of the epigenome and the environmental and behavioral contributions of a second parent to an offspring's development are being examined as well. Understanding how and why that role tends to be downplayed or ignored in the literature helps us to understand the problem of making maternal bodies the locus of responsibility. For example, advanced maternal age is considered a significant risk factor with regard to pregnancy and the health of the fetus. Individuals who become pregnant over the age of 35 are labelled as high risk for a number of complications, and depending on where they live and what is available to them economically, they may be pointed toward significantly more prenatal testing for this reason. However, an emerging feminist critique of the responsibility discourse in epigenetics notes a lack of similar conversations surrounding advanced paternal age, despite indications that the methylation patterns of older men's sperm may lead to increased health

risk as well (for example, for some neuropsychiatric conditions) (Hens 2017). The focus on women's age at conception then, in light of epigenetic knowledge, may be not only ageist, but sexist. Kristien Hens, in "The Ethics of Postponed Fatherhood", argues that the hidden message of ignoring men's contribution to the epigenetic health of their offspring here is to entrench this idea that the mother is solely responsible for the health of their offspring, increasing the amount of pressure on mothers for "good" decision-making around their environment and exposures (Hens 2017).

Assumptions about both nature and nurture, science and social sciences, then cycle into a feedback loop with regard to epigenetics and maternity. Historical and social context leads us to assume a specific kind of relationship between mother and fetus or mother and child, so attention becomes even more focused on maternal responsibility. Mother is conceived in relation to the child as an oversized source of developmental changes, both in humans and in the anthropomorphized rats in animal studies. In centering "nurture" primarily in the maternal body, not only are the implications of the mother-figure over-emphasized, but its negative image is also ignored. Fathers – or, more broadly, any other individuals coming into frequent contact with a child – have similar capacity to provide or withhold nurturing behaviours.

What next?

In this chapter, I have examined the origins of epigenetics not only as a scientific field, but as a contributor to pathologization discourse. I have raised the concern that in overvaluing molecular evidence for interactions between bodies and their environments while undervaluing existing research on these interactions from the social sciences perspective, we oversimplify the immense complexity of human health as a qualifiable, rather than merely quantifiable, experience. Moreover, we risk setting the wrong priorities as the field progresses. Despite optimism for

epigenetics' use in policy-making, Dupras and Ravitsky, situating molecularization and biomedicalization within Foucault's (neo-liberal) "regime of truth", have raised concerns that

> the impetus to create new biomedical interventions to manipulate and reverse epigenetic variants is likely to garner more attention than effective social and public health interventions and therefore also to garner a greater share of limited public resources (Dupras and Ravitsky 2016).

One potential re-correct for the field, as I will discuss in the following chapter, is for scientists and bioethicists working in the field of epigenetics to draw on insights from disability studies and critical disability theory, as an area of research that has already given immense thought to problems of pathologization and embodiment within the field of biomedicine. Such collaborations between different areas have already been raised as a particularly useful methodology for the epigenetics arena:

> In particular, the co-productionist framework will allow to unpack how the processes of gathering, standardizing and certifying epigenetic evidence will align with political, legal and economic rationalities in bringing about new settlements (or possibly reinforcing existing ones) across some of the most persistent dichotomies that structure our reflection on the human experience: normal versus pathological (or enhanced), safe versus dangerous, natural versus artificial, individual versus collective (Meloni and Testa 2014).

These collaborations are necessary in order to understand the full social, philosophical, and policy impacts of this emerging science. Next, I discuss the contribution that disability theory can make in this conversation.

Chapter 2 – Epigenetics through a Disability Theory Lens

Varieties, Structural defects, Heterotaxy, and Monstrosities: monstrosities being complex anomalies, very serious, making the performance of one or more functions impossible or difficult, or producing in the individuals so affected a defect in structure very different from that ordinarily found in their species (Canguilhem 1989, 134)

Introduction

In chapter one, I examined epigenetics as an emerging field and considered some of the historical assumptions that it is feeding off of in the creation or continuation of harmful conflation of certain kinds of bodies with epigenetic "deficits" and pathologizing variations in the epigenome. In this second chapter, I will introduce concepts from disability theory, previously unexplored in the epigenetics context, in order to produce a new method for critiquing these emerging narratives. In order to do this, I will first provide a brief explanation of disability theory, and some of the dominant models it employs. I will then show how these models can be used to demonstrate gaps in the field of epigenetics bioethical analysis as it currently stands, focusing particularly on the areas of embodiment of disability and the harms of applying morally value-laden terminology to bodily differences. I will elaborate on the connection between epigenetics discourse and preconceived notions of embodiment, heritability, and responsibility, and propose critical disability theory as a way of dismantling the presumptions at work in current epigenetic discourse and of building alternative frameworks. Finally, I will address the importance of including disabled voices in epigenetics analysis, pointing not just to the pragmatic, but the epistemic harm that results from their exclusion. This will set up the approach taken in my final chapter, where I employ the example of narratives surrounding causal and curative approaches to autism epigenetics in order to demonstrate on a concrete level the harms that I have discussed in chapters one and two.

Disability Studies and Critical Theory

To begin, it is important to make a distinction between disability studies, as a field, and critical disability theory, as both a methodology and a form of praxis. Disability studies as a field emerged largely in the 1990s, and is the academic study of disability. It is situated alongside other traditional forms of study as an interdisciplinary field; it is broadly concerned with unpacking the medical model approach to disability and engaging scholars in a social model (both of which will be defined in detail below) (Mollow 2017). Critical disability theory, by contrast, refers to a collection of interdisciplinary theoretical *approaches,* with the intention of analyzing disability as "a cultural, historical, relative, social, and political phenomenon" (SEP). As is the case with other forms of critical theory, the intention is not for insights gleaned from critical disability theory to remain in the classroom, but rather to be explicitly used for activism and advocacy. Critical theories as a whole engage with "historical strategies of domination and liberation"; as we discussed in chapter one with Foucault, oppression and power shape our understanding of the world in crucial ways. Critical theories, in unpacking the ways that power has shaped our knowledge production, are interested in "the social relation of the human subject to the historical conditions of production or alienation" (Kearney 1986: 2; SEP). The fundamental difference between disability studies and critical disability theory, then, lies with the latter's commitment to praxis.

Also important is that while disability studies attempt to place the disabled individual within the context of their social and environmental milieu, critical theory not only examines the history of how we arrived at our current understanding of disability constructs, but the way in which disabled identities are in continuous flux as society shifts. This is of particular interest in this modern era of frequent and rapid technological advances in biomedical sciences and

technology, where the question of human embodiment in general is called into constant question (Haraway 1985).

In this chapter, I straddle these approaches. I combine insights from the field of disability studies, particularly in their use of the medical and social models of disability, in order to situate epigenetics research and their implications for our understanding of disability (as well as implications of our understanding of disability for epigenetics research) within current biomedical practices. However, I will also reach beyond this to show the limits of even this framing, instead considering how disability concepts might shift alongside emerging and fluctuating biomedical fields. This will be of particular importance for chapter three, when I shift my focus to autism as an example of a narrative that is continuing to be damaged by epigenetics scholars. Certain kinds of differences, such as those seen in the Deaf community or in "cognitive disabilities and related communicative differences", are frequently overlooked in traditional disability studies (Burch and Kafer 2010, 212). The neurodiversity movement, which I will examine in more detail below, provides us with an excellent example of how critical disability theory methodologies can be employed not just to understand how cognitive and neurodevelopmental differences have been framed in the past, but how we might keep them from retaining old prejudices and stereotypes in light of epigenetic advances.

Models

Beginning with insights from the field of disabilities studies, I will start here by discussing the major models of disability that are currently used and contrasted: the medical and deficit models of health, in contrast with the social and neurodiversity models. I will also explain how the molecular model of the body and health championed by many epigeneticists fits in with these models. Throughout, it will be important to keep in mind that none of the models discussed here

presents a definitive way of examining the body and its experience of disability. As Jonas-Sébastien Beaudry has noted, "The strategy of developing an ontology or models of disability as a prior step to settling ethical issues regarding disabilities is highly problematic" (Beaudry 2016). First, because the definition of disability is, in places, inherently normative, and thus resists value-neutrality. Second, because once the impossibility of value-neutrality is accepted, the decision to choose one ontological model over another becomes less obvious. Instead, Beaudry urges us to consider the concept of disability as "better left ethically open-ended", or at least broad enough to encompass a multiplicity of ethical issues. As such, while I demonstrate below a clear preference for or against certain models, I acknowledge as well that each constitutes a piece of a larger, perhaps not fully definable, framework.

The Medical Model

Broadly speaking, the medical model of disability is the one most commonly known; it is the method of evaluating health that is most commonly employed in standard medical practices. The medical model is one that essentially locates the disability within the individual; to classify someone as disabled under this model is to make a comment on the person's physical body (Marks 1997). For example, under this model, an individual who utilizes a wheelchair for mobility assistance is considered to be disabled due to deficits in their ability to walk unassisted. This is a particularly normative view: people are considered disabled because they function differently from the perceived "normal" person (Roush and Sharby 2011). The focus of defining and diagnosing disability under the medical model tends to be so that modifications can occur to the body to then remove the disability label. Under this model, disability is often seen as the defining characteristic of the disabled individual, and there is a presumption that the individual will be inclined toward seeking a cure.

We can generally locate the shift to the medical model of disability discourse in the shift of power toward medical professionals discussed in chapter one. In particular, Louise Humpage argues that the medical model can be traced to the point where doctors replaced religious leaders as society's "cognitive authorities" (Humpage 2007). The replacement is, in a sense, its own kind of faith-based approach; "the medicine with which we are familiar is a project of a modernist faith in the ability of reason and rationality to drive continued technological and social advancement" (Scully, 51). At this same time we see a continued reductionism of the body, moving from an overall concept of health to one that focuses on defining illness and injuries at the most micro possible level, rendering a complete understanding of health more and more difficult for a person who does not have medical training (Brittain 2004). As Scully notes, "modern medicine committed itself to conceptual models that could only be used with confidence by experts, and which excluded the sick or disabled person's own comprehension of his/her state" (Scully 2002, 49). With an understanding of human health centered on specialized medical knowledge, the conception of disability put forth by the medical field becomes central; this conception largely situates disability as a biological fact (Haegele and Hodge 2016, Brittain 2004).

Among the consequences of the medical model of disability are an increase in "cure" rhetoric, where the focus of medicine becomes the rectification of disability and the movement of the disabled body back along a biological spectrum as close to the centre (the "normal" body) as possible. At its worst, we see this played out in eugenics rhetoric, where the disabled body is so undervalued that it becomes preferable to prevent or end the existence of disability (and thus, disabled individuals) than to put work into accommodations. We have already begun to see the role of cure rhetoric in epigenetics research, the science side of which is largely situated within the medical model. This is particularly evident in the framing of epigenetic "deficits", variants that are

seen as causal factors in the development of disability. These are targeted as mechanisms for curing disease, both through epigenetic-specific treatments and through attribution of moral responsibility to environments that perpetuate these epigenetic shifts. We are also beginning to see the rise of "treatments" offering to optimize one's epigenome; the fact that the science does not yet bear out these possibilities is in some ways beside the point. As it currently stands in the public understanding, the epigenome becomes another standard by which to separate the healthy and the unwell.

The Social Model

In contrast with the medical model of disability, the social model of disability is one that locates disability in the oppressive social environment (Marks 1997). Broadly speaking, the social model "contests that it is society that imposes disability on individuals with impairments" (Haegele and Hodge 2016). Instead, the social model examines the barriers and attitudes facing individuals with bodies outside of the norm, that result in discrimination or social exclusion for disabled individuals, and consider these social factors to themselves be a (or the) source of an individual's disability. For example, unlike in our medical model example above, an individual who uses a wheelchair under the social model is not disabled by their differences in mobility; instead they are disabled because we live in a society that is not built for accessibility for wheelchair users. In practice, this simplified version of the social model is often used to engage in political arguments; there are implications for areas such as funding, for instance, where a social model privileges resources being used to provide equitable access to society, rather than toward curative approaches that would render such access unnecessary.

On a more complex look, the social model is not a single model but a series of models (Haegele and Hodge 2016). Per Mitra, there are nine different versions of the social model

including "(a) the social model of the United Kingdom, (b) the oppressed minority model, (c) the social constructionist version of the United States, (d) the impairment version, (e) the independent living version, (f) the postmodern version, (g) the continuum version, (h) the human variation version, and (i) the discrimination version." (Haegele and Hodge 2016, Mitra 2006). These models exist primarily within the realm of academic theory (and sometimes politics); unlike the medical model, which is ubiquitous, the social model is employed more as a normative framework, in the hopes of generating a shift away from the medical approach.

The social model has its own critics, even within the field of disability studies. In particular, it has been criticized for its over-zealous application; Shakespeare has argued that it is best perceived as a political tool to be employed within an inadequate framework, rather than a model unto itself. (Shakespeare 2004). Others have critiqued its failure to address impairment "as an observable attribute of an individual that is an essential aspect of their lived experience". (Haegele and Hodge 2016, Palmer and Harley 2012); this is an argument to which I will return below in outlining the neurodiversity model. Finally, some critical disability theorists have expressed concerns that the social model, and traditional disability studies more broadly, have failed to adequately capture the real concern of pain as a debilitating factor in disability (Mollow 2017). Nevertheless, in focusing our understanding of disability on the lived experience of the disabled individual rather than the body alone, the social model does provide a good starting point – or at least parallel – for criticisms of epigenetics research that reduces genetic and environmental interactions to the molecular.

The Neurodiversity Model

Although not explicitly a disability studies model, the neurodiversity model provides another window through which disability – cognitive and neurodevelopmental in particular – can

be viewed. Contrasted with the medical model's approach, which views cognitive and intellectual differences as deficits, the neurodiversity movement has sought to embrace cognitive differences as part of a necessary and positive spectrum of human experience. Although the movement originated in the late 1990s as a non-pathologizing approach to variation in the human brain, it became more ubiquitously known outside autism circles via Steve Silberman's 2016 text *Neurotribes* (notably, sociologist Judy Singer, who is credited with developing the term, is an autistic woman; as I will note further in chapter three, exclusion of autistic women from the medical discourse is common) (Singer 1999). Neurodiversity views autism through the lens of the social model rather than medical model of disability, where the disabling factors stem primarily from a lack of resources and acceptance rather than an inherent deficit (Silberman 2016). Silberman defines neurodiversity as:

> the notion that conditions like autism, dyslexia, and attention deficit/hyperactivity disorder (ADHD) should be regarded as naturally occurring cognitive variations with distinctive strengths that have contributed to the evolution of technology and culture rather than mere checklists of deficits and dysfunctions (Silberman 2015, 16).

Contrasted with the social model, the neurodiversity model is nevertheless more open to identifying and mitigating weakness or harms that may accompany neurodivergence. For instance, activists in these areas have called for research into health challenges often experienced by autistic individuals, such as epilepsy and gastrointestinal distress. The focus is on depathologizing the social aspects of autism, which are frequently the primary focus of researchers under the medical model.

As it has developed, the neurodiversity movement has come to consider autism as a distinct community and culture (Bumiller 2008, Bowker and Tuffin 2002), and has emerged as a counter-narrative to the medical and deficit model approaches to autism, one that does not rely on physicians for its definition. More recently, the concept of "neuroqueer" – a project of

"disidentification" that "rejects both oppressive dominant and counterculture identities that perpetuate destructive medical model discourses of progress and cure" – has emerged, bringing with it another layer of analysis that examines the intersections of disability, gender, and sexuality (Egner 2019, Richter 2016). As such, the growing neurodiversity and neuroqueer movements have seen more space for self-diagnosis and self-identification with autism.

The neurodiversity model, perhaps more so than the social model, enables us to engage with epigenetics discourse by situating the disabled individual within their broader context, and to consider how certain epigenetic differences that might be perceived as harmful or disabling in epigenetics research are in fact more reflective of environmental adaptations that *become* advantageous or disadvantageous under such conditions. Within the neurodiversity movement, for instance, it is not uncommon to see discussion of features of autism commonly considered clinically problematic – for example, challenges in navigating everyday sensory environments – repurposed and redefined as autistic strengths, such as the attention to detail that may accompany the intensified sensory experience. Looking back to chapter one, where I discussed the concept of the uterus under adverse social and environmental conditions being framed as a "metabolic ghetto", the neurodiversity model give us space to consider how the fetus under such condition is actually responding impressively to environmental challenges, remaking itself to adapt to its surroundings.

The Molecular Model

Finally, although it is not considered a disability theory model unto itself, for the purposes of understanding how epigenetics fits into broader conceptions of health, it is necessary to consider the emerging molecular model of biomedicine. In "A postmodern disorder: moral encounters with molecular models of disability", Jackie Leach Scully reminds us that "biomedicine presumes there

is only one way of knowing the body" in modern medicine, this way has been increasingly microscopic (Scully 2002, 54). Although often framed in terms of the influence genetic medicine has on our understanding of the body as a whole, the molecular model is in fact "a far more radical reduction of the whole concept of disease than that proposed by genetics" (Scully 2002, 50). While the *genetic* model, a sub-category of the medical model, views human disability as being primarily associated with "aberrant" or "defective" genes, it restricts itself to understanding how the relationship between gene and illness emerges. Scully refers to the molecular model as the *meta-narrative* that underpins modern medicine; unlike the genetic model, in the molecular model "the production of abnormality, the complete pathological process, can be successfully explained by molecular interactions without invoking any other factors" (Scully 2002, 51). Like the genetic model, it is not a standalone approach, but rather a sub-category of the medical model. It is in this model that epigenetics research has thus far largely resided, despite hopes that it would in fact raise the study of genetics out of the molecular model altogether and place it within a broader social framework. Given that "[m]edicine is the business of reconstituting normality and health", what the situation of epigenetics within the molecular model serves to do is to offer up epigenetics not only as a piece of understanding human health, but instead, as a suggestion that by controlling external environments, we can render the body at a molecular level back to an optimal form (Scully 2002, 48).

Bioethicists working in the field have already raised concerns about this kind of about neoreductionism in epigenetics (Meloni and Testa 2014). While epigenetics did not invent the idea of diminishing the human story and experience down to the cellular level and below, Niewöhner has argued that epigenetics nevertheless furthers the trend toward a 'molecularisation of biography' (Niewohner 2011), where the individual experience of their own environment and

behaviours, their own narrative, are reduced to its impact on gene expression. Epigenetics as a whole straddles this tension between macro and micro narratives; environmental epigenetics in particular has "genealogical" origins in the study of epidemiology and the social determinants of health and the developmental origins of health and disease, addressing "hypotheses accepted as legitimate within these fields but now at a molecular level" (Lewis and Thomson 2019). While this is viewed as the "promise" of epigenetics – the provision of "real" biological data to back up population-wide and social sciences narratives – in doing so, it appears that the social in fact has ended up diminished to the biological. Even though "in epigenetics biomedical knowledge and the social structures of parenting, gender and family life mix in a range of ways", contributions by social scientists to our biomedical knowledge have been viewed as less critical than the contributions from molecular sciences to the social (Pickersgill et al. 2013, 437).

One thing that critical disability theory methodologies and frameworks enable us to do is to interrogate this trend toward biological neoreductionism in epigenetics, arguing instead that epigenetics should push us to consider the value of body *as experienced*. As Scully has argued, "the lived experience of a specific embodiment affects the structures of imagination and interpretation that people use in moral perception and evaluation" (Scully 2003). Rather than an approach that views epigenetic markers only as molecular evidence of experiences, a disability or critical disability theory-informed approach that recognizes the body as embedded in its macro environment could contribute to epigenetics becoming part of a more holistic understanding of health.

Embodiment, or Disability in Milieu

Viewed through a lens of optimism, then, epigenetics can be harnessed to provide us with exactly the kind of counter-narrative to genetics reductionism championed by critical disability

theories. In situating the body firmly in its social and environmental milieus, epigenetics renders it impossible to forget that examining body *qua* body without attention to its surroundings is no more than a theoretical exercise. Real advances in medicine and our understanding of the body require that we take the body as-is: messy, complex, and embedded. From its side, disability theory, in situating the body within its social and physical environment, can assist us by providing a mechanism and methodology to interrogate this question of the body's positioning within "space and in the milieu of expectations" (Garland-Thomson 2017, 20).

Challenging environments produce bodies that react to those environments; an adverse environment does not result in an adverse body. Rather, it produces a body optimally created to survive those circumstances; when circumstances shift and the body does not keep up, this does not mean that a "bad body" is the result. And yet, as Scully notes in her discussion of the conflict between valuing bodies as inherently good, and recognition of the suffering that may accompany them, "the human experience of embodiment is often, perhaps always, profoundly ambivalent" (Scully 2003, 279):

> In its entirely correct desire to reclaim bodily experience as a site of goodness, what contemporary body theology absolutely must not do is to sideline the equally real experience of the body that is limited in time and space. It must not turn away from the ageing body, or the body immobilized, breathless, convulsing or demented. The 'good' body and the 'bad' body coexist, they are one flesh-and, as we have seen, our understanding of good and bad may be modified by circumstances (Scully 2003, 279).

Both critical and traditional disability theories can also help us to deal with the dual concerns that, 1) the environment can have real impacts on bodies at the molecular level, and 2) the resulting body does not need to be assigned a moral value. Consider two different framings in the question of epigenetic changes that result from a lack of sufficient prenatal care. While the ultimate goal of improved policies surrounding access to prenatal care might be shared in both these cases, a framing that establishes mothers under those conditions as "metabolic ghettos" resulting in

epigenetically inferior fetuses is fundamentally different from one that recognizes that external social and environmental factors during gestation can impact health outcomes later in life. We can use lessons from social and neurodiversity models to deconstruct the conflict between acknowledging the need for access to nurturing environments (both social and physical) and the need to avoid demonizing bodies that represent the outcome of environmental adversity.

Donna Haraway explored this delicate balance between body and the tools used to enhance the body's function in her work on the concept of the cyborg as a rejection of limitations and boundaries of both the body and the broader self, asking "Why should our bodies end at the skin, or include at best other beings encapsulated by skin?" (Haraway 1985, 33). Although epigenetics thus far constitutes mainly a method of understanding the body, interest in epigenetic enhancement and epigenetic therapy suggests that epigenetics may be used not just to better understand these connections and to make accompanying shifts in environment or consumption, but to manipulate the epigenome more directly (Kelly and Issa 2017). Shildrick, in discussing Haraway from a disability perspective, notes that while critical disability theorists have raised concerns about problematic use of technology for restoration of the body to a normatively "neutral" point, in modern terms "rehabilitation to normative practice or appearance is no longer the point; instead, the lived experience of disability generates its own specific possibilities that both limit and extend the performativity of the embodied self" (Shildrick 2015).

Shildrick considers "not just technological aspects such as prostheses but an array of materials, locations, and spaces that might all be called technics", which "encompass an arena of action far in excess of two or more intersecting bodies, although what is always figured is the instantiation of particular bodies" (Shildrick 2015). Epigenetics similarly asks that we expand our understanding of the body beyond the contained flesh and into the surrounding environment – not

just at the micro level presupposed by molecular sciences, but at the macro level envisioned by social and environmental epigeneticists. Epigenetics, like disability theory, pushes the body beyond the limitations of skin to recognize the interdependence of body and all that touches on it. The womb in particular becomes much more than a mere fetal environment, and instead an extension of the fetus, providing not just a space to gestate but a milieu for the genome to learn to express itself.

Epigenetics and disability theory share an ontological question about embodiment: where do we rightfully place the boundaries of a person with regard to their environment? Under the medical model, both curative approaches and assistive technology have made promises to disabled people that they will see their disabilities diminish. However, for many disabled individuals, these "solutions" may act as a modifier to their disability that nevertheless does not touch on disability as identity, or may make changes to their identity as a disabled person in a way that is shifting rather than static. As Ingunn Moser notes, being disabled

> is not something one is but something one becomes, and, further, […] disability is enacted and ordered in situated and quite specific ways. The question, then, is how people become, and are made, disabled – and, in particular, what role technologies and other material arrangements play in enabling and or disabling interactions. (Moser 2006)

Technological solutions may blur the line between the disabled person and their tools and environment, such as through the use of cochlear implants. The body remains the same, but the boundary between person and their surroundings shifts in ways that may modify self-understanding. Epigenetics can similarly serve to blur the boundaries between person and environment, if harnessed to do so. Lewis and Thomas, for instance, argue that:

> In the twenty-first century, the body that is emerging from neuroscience and epigenetics is embedded within and shaped by its milieu. This body is further shaped by its passage into public, legal and political spheres. (Lewis and Thomson 2019)

Elsewhere, epigenetics ELSI scholars argue that "recent critiques of the separate worlds of biology

and culture, and the rise in epigenetics, provide new opportunities for expanding theoretical concepts like [Bourdieu's] *habitus*" (Warin et al. 2016, 53). In discussing the challenge of navigating the medical and moral, Warin suggests the necessity of doing so by developing a concept of "*biohabitus* – reconfiguring how social and biological environments interact across the life course, and may be transmitted and transformed intergenerationally" (Warin et al. 2016).

The Good, the Bad, and the Epigenetically Monstrous

The medical model of disability presents a number of difficulties for understanding the disabled experience. Perhaps most pressingly, however, it engages not only in a *taxonomical* question – who is "normal", and who is different enough to require intervention – but a *moral* one. Although epigenetics research is still in its infancy, there is already evidence that the attribution of moral weight to the body and health is a continuing trend. Research in environmental epigenetics in particular has shown a trend of framing particular environments not just of influencing epigenetic development in one direction or another, but explicitly engaging in epigenetic "harm"; legal scholars in particular have seized on this concept as a new way of framing civil liability evidence for environmental torts (Kabasenche and Skinner 2014, Wiener 2010). Other papers employ the language of epigenetic "deficits" or epigenetic "defects", both of which contribute to the presumption of an ideal epigenome, like the ideal, non-disabled body, toward which biomedical sciences might aspire (Holliday 1987, Lee et al. 2013).

In part because of framing like this, Scully has argued that disability as a class is different from other oppressed classes (based on gender, race, etc.), because there is nothing *inherently* disadvantageous about those (Scully 2003). This represents part of the challenge discussed above raised by the social model of disability; there is a difficult line to tread where by disability, we already *mean* a harmful deviation from the norm; by contrast to race or gender designations,

> the label that accompanies the ontological category of 'disability' is already in itself an evaluation, as we can see if we think about what remains outside it: members of the category 'able-bodied' or 'not disabled' or 'non-impaired' or even 'normal'. (Scully 2003)

Some of this is the result of the way that particular concepts are employed across modern ethical and bioethical theory; Scully particularly mentions traditional features of moral theory, such as autonomy and independence. Critical theories – in particular, disability theorists and feminist ethicists – have long critiqued these concepts, which "fail to reflect the lives of those whose physical vulnerabilities make autonomy and independence, as commonly understood, meaningless" (Scully 2003). Implicitly embedded in the curative approach to the medical model is the idea that when a body becomes disabled, it has diminished in value and must again be rendered whole. This curative approach is premised in part on economic considerations; not only are disability accommodations considered costly, but a disabled body is one that is literally considered to produce less value under a capitalist system.

However, critiquing value judgements in the realm of disability and disease is complicated when we recognize the very real suffering that ill health can cause; even under the social model of disability, there remains a difference between disability and unhealthiness (Wendell 1996). This raises further questions about the medical model's drive to end or prevent disabling differences; as disability theorist Susan Wendell writes, in discussing the disabling quality of her chronic illness: "If we value the differences of people with disabilities (as I do), what implications does that have for efforts to prevent or cure disabilities?" (Wendell 1996, 8). Although it is a subject of some debate, disability theorists have not demarcated (and indeed, will never be able to fully demarcate) a clear line between embraceable human differences and debilitating disease. However, what is clear is that the medical model's blanket approach of naming *all* differences as deficits is just as clearly harmful, and it is already apparent in epigenetics research. A Google Scholar search

for the phrase “epigenetic defects” yields 4,800 results as of 20 April 2020, including papers linking variations in the epigenome to not only to illnesses like cancer or heart disease, but to conditions like “mental retardation”, aging, autism, deafness, and dwarfism (Kramer and van Bokhoven 2009, Holliday 1993, Issa 2014, Miyake et al. 2012, Annavarapu 2019). The more value-neutral “epigenetic difference”, by contrast, provides 197 results. Of these, 50 refer to twin studies, and 112 are at least partly premised on animal models (Demir and Demir 2018, Lazar et al. 2018)

The line between what is a variation in the human epigenome and what is an epigenomic “defect”, is not a question that has been thus far engaged with fully in epigenetic ELSI scholarship (Dupras, Saulnier, and Joly 2019). So long as epigenetics literature participates in the idea that some bodies are epigenetically “inferior”, it is not only inheriting the problems of the medical model and its accompanying pathologization, but actively contributing to them; “Biomedical science profoundly shapes our assumptions about what a normal body is, how it should behave, *when a bodily change is threatening* and what the natural limitations of the body are” (Scully 2002, 53) (emphasis mine). The question of what constitutes a difference and what constitutes an impairment is not a question of natural, value-neutral statistical difference, but is rather “historically specific and performative” (Tremain 2017, 115). Scully argues that no matter what, certain disabilities will always be associated with “an irreducible negative” that no amount of social transformation can rectify (Scully 2003). However, I would argue that, to a degree, the power does lie with the biomedical sciences to redefine these valuations.

Returning to Canguilhem, in the taxonomy of human variation, differences might be considered monstrosities not only if they are “complex anomalies, very serious, making the performance of one or more functions impossible or difficult”, but if they “[produce] in the

individuals so affected a defect in structure very different from that ordinarily found in their species" (Canguilhem 1989, 134). The question of what becomes epigenetically defective – or, indeed, epigenetically monstrous – hinges partly as well on the "human factor" in producing epigenetic variants. When a person is viewed as personally responsible for a change in their health, an element of moral accountability is cast over the development of disability (Minkler and Behavior 1999). This rhetoric has developed strongly in epigenetics research; because of the potential for human behaviour (both social and in terms of "avoidable" exposures) to impact epigenetic variation, ELSI scholars have warned of a potential rise in attribution of moral responsibility to unhealthy or disabled individuals based on an examination of their epigenome (Hedlund 2012). This potential for a rise in individual responsibility is intensified by the heritability of epigenomic variants, as exposures creating epigenetic modifications may not only impact the individual, but their future children. As I will demonstrate in the next chapter, these narratives have the potential to cause real harm.

What Next?

In this chapter, I have introduced various models and frameworks emerging both from disability studies and from critical disability theory, and have examined the ontological and ethical implications of applying these theories to the study of epigenetics. As critical theory, however, invites us to engage not only in theorizing, but in praxis, it remains for us to consider the real-world effects of employing epigenetics as a frame of reference for human experience in the absence of disability critique. As such, in the following chapter, I will take the concepts outlined in chapters one and two and employ them in the example of the autistic experience, in order to demonstrate how bad philosophy becomes in reality bad practice, and even bad medicine. I will apply the disability theory lens to the history of autism narratives since they first emerged, and see how

language of deficits and blame have come to be built into the epigenetic approach to examining autism. In particular, I will focus on how the story told by this bioreductionist and value-laden approach to epigenetics causes real pragmatic and epistemic harm to autistic individuals, and argue that engagement with disability discourse as shown here is critical to prevention of real-world harm.

Chapter 3: Epigenetics and Autism: An Epistemic Problem

"In the final analysis it is the patients who most often decide – and from very different points of view – whether they are no longer normal or whether they have returned to normality." (Canguilhem 1989, 119)

Introduction

In the previous chapters, I have laid out the historical grounding that has led us to modern-day epigenetics science, as well as modern-day rhetoric around the hype and hope for epigenetics research. I have also explained the ways in which disability theory points to flaws in this approach, and addresses concerns around reductionism of the social to the molecular. In this chapter, I will give a specific example of how these concerns are playing out in real time in autism research, using the autistic community and their experiences with value-laden research as a proof-of-concept to demonstrate how autistic people are harmed in a real way, and in particular, suffer an epistemic injustice when epigenetics researchers fail to take into account the context of their findings. Starting from a framework that presupposes the value of a neurodiverse population, I argue that autistic individuals suffer from the application of normative values that have accompanied the medical model/epigenetic "deficit" approach to considering cognitive well-being, focusing on how these narratives are acted out upon the individual.

To do this, first, I will explain the history of harmful and ableist rhetoric in autism research, paying particularly close attention to the places where the language of *responsibility* and *blame* have surfaced, especially in relation to the role of mothers in the creation of an autistic child. I will highlight how maternal bodies are viewed in epigenetics literature more generally, and consider some emerging ideas about personal responsibility for one's epigenome. Then, I will examine the epigenetics research arising in autism fields, and consider how problematic historical conceptions of the relationship between autistic individuals and blame and responsibility and mothers is resurfacing in epigenetics research, and discuss the implications of this for autistic individuals and

their families. Finally, I will argue that the harms that emerge from this framing in epigenetics research harm autistic individuals not only by increasing stigmatization and pathologization and redirecting resources away from the autistic community's real needs, but, in hijacking the autistic narrative and contributing to an inaccurate genealogy of autism, harm autistic individuals epistemically.

Autism History

Although awareness of autism has increased in recent years, a great deal of misinformation persists. In strictly clinical terms, under the medical model, autism is defined as a developmental disorder of complex, largely unknown origins. While the term "autistic" was first coined as an adjective in psychiatry in the 1910s, most historical accounts attribute our current understanding of autism to the two Austrian doctors who were the first to publish on the subject: Hans Asperger, and Leo Kanner. Notably, Grunya Efimovna Sukhareva, a young female doctor working in Moscow in the 1920s, is not usually mentioned, despite having published an account of six young patients in 1926 (Zeldovich 2018). Since that time, autism studies have undergone a number of changes, shifting from a perception of autism as largely environmentally-based – the result of insufficiently nurturing so-called "refrigerator mothers" – to the modern idea of autism as resulting from a combination of environmental and genetic – or epigenetic – causes (Kroncke, Willard, and Huckabee 2016).

The diagnostic criteria for autism continue to evolve, albeit contentiously. The DSM5, the 2013 update to the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, fundamentally redefined autism, replacing three separate diagnoses of Autistic Disorder, Asperger's Disorder and Pervasive Developmental Disorder – Not Otherwise Specified (PDD-NOS) with the singular diagnosis of Autism Spectrum Disorder (King et al. 2014). Much

of the controversy over this decision concerned the possibility that individuals eligible for much-needed social and medical services under the previous definition might lose their access to these services, as well as a loss of nuance in the understanding of different manifestations of autism, particularly through the under-identification of individuals who would have previously been classified as experiencing PDD-NOS (Mayes, Black, and Tierney 2013).

Under a strictly medical model, which, as discussed in chapter two, focuses on the physical identification of diseases and disorders with an emphasis on curative approaches, autism is largely identified by perceived deficits in the cognitive functioning of autistic individuals. Under the new DSM, diagnostic criteria for the autism spectrum are divided into two categories: first, "Persistent deficits in social communication and social interaction across multiple contexts" (including social-emotional reciprocity; nonverbal communicative behaviors used for social interaction; and developing, maintaining, and understanding relationships); and second, "restricted, repetitive patterns of behavior, interests, or activities" (including stereotyped or repetitive motor movements, use of objects, or speech; insistence on sameness, inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behavior; highly restricted, fixated interests that are abnormal in intensity or focus; and hyper- or hyporeactivity to sensory input or unusual interest in sensory aspects of the environment) (American Psychiatric Association 2013). This description of autism as a collection of deficits is controversial in the autism community, and is seen as both resulting from and contributing to the exclusion of autistic voices from the discourse surrounding autism (Dinishak 2016).

There is a longstanding history of shame and blame involved in both autism research and media reporting on autism, beginning with the causal assumptions made by early autism researchers. The resulting harms have been profound. As I will demonstrate below, incautious

epigenetic autism research exacerbates existing harms to autistic individuals and their families, including high rates of depression, anxiety, and suicide, as well as caregiver abuse spanning from potentially fatal curative attempts and infanticide. These harms have been particularly pronounced for autistic women, who have been all but completely excluded from autism narratives. Hans Asperger originally thought that no women or girls were affected by the syndrome he described in Autistic Pyschopathy in Childhood (1944), although later clinical evidence caused him to revise this thinking (Asperger and Frith 1991). Similarly, in Leo Kanner's 1943 study of a small group of children with autism, there were four times as many boys as girls (Haney 2016). The gendered accounting of autism that existed from these earliest clinical descriptions was later exacerbated and cemented with the publication of autism researcher Simon Baron-Cohen's "extreme male brain theory" (Baron-Cohen 2002). Baron-Cohen sorted thinking styles into two gendered types - feminized "empathizing" and masculinized "systematizing" - and attributed the most systematic brains to those on the autism spectrum (Baron-Cohen 2010). Feminist scholars in particular have argued that this "essentialist" version of autism reconstructs gender stereotypes while masquerading as science (Bumiller 2008), and indeed, even Baron-Cohen now acknowledges the diagnostic disparity caused by researchers ignoring the "female phenotype" of autism (although he neglects to note his role in the creation of this problem) (Lai and Baron-Cohen 2015). Autistic history is filled with examples of the harms that result from entrenched misunderstandings about the nature of the thing becoming an intractable part of the narrative.

There are real, tangible impacts from these harmful stories. Many autistic individuals experience significant mental health challenges – particularly involving depression and social anxiety – and for women in particular, physicians may neglect to further pursue the underlying causes of these diagnoses (while social anxiety can form a part of the autistic experience, and

depression is often present, particularly in undiagnosed autistic adults, without the key understanding of autism, interventions for these issues will be less effective) (Halladay et al. 2015). Because autism research, working under the medical model, has set its focus on prevention and cures, the results of this research have often failed to benefit autistic individuals themselves. The therapies that have been developed for use with autistic children are structured with the goal of reducing the *appearance* of overt autistic behaviours, rather than providing relief from navigating the neurotypical world. The most widely-used of these, applied behavioural therapy (ABA), has faced intense criticism from within the neurodiversity movement for its goal of creating more compliant, rather than healthier and happier, autistic individuals; some autistic individuals who have experienced ABA have likened it to torture (Croen et al. 2015). Research into the genetic and epigenetic root causes of autism have also been met with skepticism and concern; they raise concerns amongst autistic individuals that causal research will be used for cure rhetoric, leading to prenatal tests or treatments that could be used to diminish the autistic population (Bumiller 2009).

None of this is to argue that there is not an interest from the autism community in research that would further unpack the differences between autistic individuals and their neurotypical counterparts. There are also a number of medical and mental health conditions that are correlated with autism – although the question of whether they share a causal basis with autism or result from discrimination is a difficult one – and treatments for these are of interest to many autistic individuals (Croen et al. 2015). Epilepsy, sleep difficulties, and gastrointestinal issues such as IBS and gluten-intolerance, and auto-immune diseases affect quality of life for many autistic individuals. Autism advocates have voiced worries that research focusing on the causal roots of autism, rather than on these health concerns, is another case of the priorities and needs of the

autism community going ignored (Pellicano, Dinsmore, and Charman 2014). Anxiety and depression, which are also frequently co-diagnosed with autism, may stem from similar causal factors, or may be the result of stigmatization and discrimination. Finally, schizophrenic disorders, obsessive-compulsive disorder, bipolar disorder, and attention-deficit disorders also appear frequently in autistic individuals, and are themselves stigmatized conditions (Croen et al. 2015).

It is clear, then, that the history of autism research shares some of the problematic features of other advances in biomedicine discussed in the previous two chapters. In particular, though, autism research shares a feature with epigenetics research that leads to the concern that epigenetics research *about* autism may compound these past harms; namely, a tendency to move beyond medical facts to concern itself with assigning responsibility and blame for the differences that autistic people represent. Below, I will explain some of this history.

Refrigerator Mothers

One of the biggest and earliest examples of this intersection motherhood and blame in autism is that of "refrigerator mother" rhetoric. Of our nature-nurture debate described in chapter one, this is the example stemming most clearly from the "nurture" side of the equation. Originating in 1943 with Leo Kanner's first account of Early Infantile Autism, Kanner, raised the possibility that autism was caused by emotionally cold, or "refrigerator" mothers. A 1949 article by Kanner, "Problems of Nosology and Psychodynamics of Early Infantile Autism, further described autistic children as having been "reared in emotional refrigerators" (Cohmer 2014). The contention was that autism was attributable to mothers failing to create a warm and nurturing environment for their children. This rhetoric was challenged by other psychiatrists working in the field, as well as by parents of autistic children, and by the 1970s and 1980s it was largely discredited within the field (Langan 2011). Nevertheless, the concept became tightly woven into autism narratives, and

persists in other formats. Although the rise of the biological – at the time, genetic – basis for autism in the 70s and 80s helped to dispel some of this mythology, new epigenetic theories about the contribution of the uterine environment to the development of autism threaten to bring this theory back into the spotlight, albeit in a new format.

Research into the impact of the "refrigerator mother" theory has largely focused on the way that the discourse has harmed mothers of autistic children (Weusten 2011). Autistic individuals, however, have continued to experience harm from this theory as well, in part due to its contribution to the idea that autism might be "curable" and to therapies that focus on diminishing signs of autism in the individual rather than helping the autistic individual navigate their world as-is. In 1983, for instance, biologist Niko Tinbergen and his wife published their psychogenic theory of autism, arguing that it can be cured through "holding therapy". Elsewhere, the refrigerator mother discourse continues to be reflected in the idea of autistic children as representative of maternal failure – which, indeed, co-signs the idea that the autistic child is, in and of themselves, a failure (Osteen 2008).

Vaccination Controversy

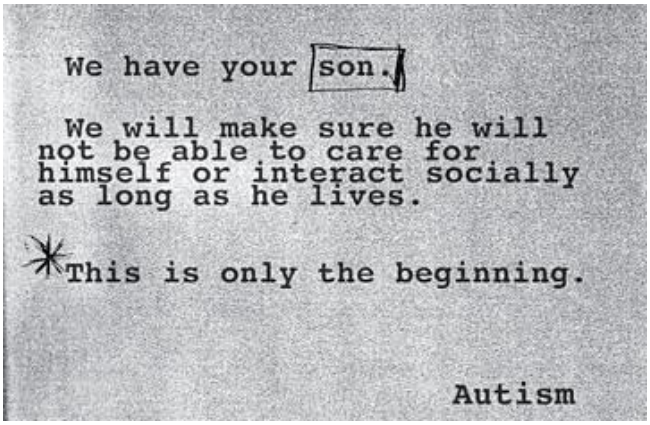
A large-scale misconception around autism that helps to demonstrate the connection between harmful responsibility narratives and bad science is the autism and vaccination controversy. In 1998, Andrew Wakefield published in The Lancet a paper linking the MMR (Mumps, Measles, Rubella) vaccine to development of autism (Wakefield 1999, Wakefield et al. 1998). Using an n of 12, an uncontrolled and unethical study design, and providing speculative results, Wakefield claimed a link between vaccination and behavioural regression (Rao and Andrade 2011). Although later epidemiological studies would soundly refute this claim, by the

time the paper was retracted in 2010, untold damage had been done (Wakefield, Murch, and Anthony 2010).

Even as it later emerged that the study had been not just unethical, but in fact a case of fraud (with the authors falsifying facts to fit their claims), vaccination rates continue to drop (Rao and Andrade 2011). As a breach of scientific ethics, this story has become a cautionary tale. As a contribution to misconceptions about autism, as well as its role in convincing parents to delay or forego the MMR vaccine for their children, the results were catastrophic. By creating a narrative link between "bad" parents (those who succumb to Big Pharma and vaccinate their children) and the development of autism, autism itself becomes a fate considered (literally, given childhood mortality rates from mumps, measles, and rubella) worse than death – one that can only be avoided through making the "right" choices as parents. Although the anti-vaccination movement does not appear to have yet broadly seized on a pseudo-scientific understanding of epigenetics to make their cases in general, one interview with prominent celebrity anti-vaxxer Jenny McCarthy sees her arguing that epigenetic predisposition to autism should be considered by pediatricians before vaccination, using the language of taking into consideration a "methylation tree" that includes depression and bipolar disorder (Frontline 2015). McCarthy, whose son was diagnosed with autism at two and a half after experiencing a series of seizures, claims to have reversed Ethan's autism through dietary changes after consultation with the organization DAN! (Defeat Autism Now!) (Frontline 2015). When asked about the impact of non-vaccination on the spread of preventable illnesses, she remained firm that "warrior moms" – implicitly coded as mothers who will do anything for their children – prefer this to having a neurodivegent child. "If you ask 99.9 percent of parents who have children with autism if we'd rather have the measles versus autism, we'd sign up for the measles" (Frontline 2015).

Awareness Campaigns

The theme of parental failure (with a focus on maternal failure) is common throughout autism research and (mis)information campaigns. In 2007, the New York University Child Study Center launched a "public service campaign" to raise awareness around childhood psyhiatric and developmental disorders. This campaign was comprised of messages, styled as ransom notes, posted throughout New York City and published in Newsweek and New York magazine (Kras 2009):



[Text is styled as a typewritten ransom note and reads: We have your son. We will make sure he will not be able to care for himself or interact socially as long as he lives. This is only the beginning. Autism] (Kras 2009)

The campaign provides an excellent example of how narratives surrounding shame and terror are harnessed in autism narratives to make it appear as though the diagnosis of autism in one's child is some kind of emergency, correlating it with a parent's worst nightmare. The implication is that without constant vigilance as a parent, autism may come along and steal your child. The campaign ran a similar advertisement for Asperger's Syndrome (now subsumed in the DSM5 under Autism Spectrum Disorders), catastrophizing the outcome of a diagnosis:

> Asperger's Syndrome: We have your son. We are destroying his ability for social interaction and driving him into a life of complete isolation. *It's up to you now* (emphasis mine) (Kras 2009).

Setting aside, for the moment, the gender bias evident in both of these advertisements: the implication of "it's up to you now" places the responsibility for "managing" their child's neurodivergent mind in the hands of the parents. The director of the centre at the time claimed to hope that the ads would encourage more diagnoses, but what these ads really did was to promote the idea that autism is something parents should be terrified of and want to avoid at all costs.

Autism Organizations

The fear-mongering shown in the New York Child Study Center advertisements has been exhibited as well by Autism Speaks, the largest autism research and advocacy group in the United States (Heilker and Yergeau 2011a). Widely condemned for its "curative" approach as well as for hate-filled rhetoric against autistic individuals (ASAN 2009), Autism Speaks ran a similar fundraising campaign that played upon parental fears around their child's diagnosis. Their video, also personifying autism as an aggressor, declared "I am Autism… I am visible in your children, but if I can help it, I am invisible to you until it's too late… I work faster than pediatric aids, cancer, and diabetes combined" (Savarese and Savarese 2009). Although these campaigns refer largely to childhood diagnosis of autism, because of gender bias in the descriptions of autistic symptomology, during a child's diagnosis is the time when many adult women receive their own diagnosis. Mothers in particularly, then, may be doubly harmed by this misinformation; first, in their ability to know their child, and second, in their ability to accurately recognize themselves in this description of autism.

Autism Speaks is particularly known for their "light it up blue" campaign, which, among other things, involves cities lighting their landmarks (such as the Sydney Opera House and the

Empire State Building) up in blue to raise awareness about autism (Dillenburger et al. 2013). Such campaigns have been met with skepticism in the autism community, as the narrative put out by charities like Autism Speaks have generated so much harm, in particular by classifying autism as a disease. Similarly, the Autism Speaks puzzle piece symbol, trademarked in blue, has become a "ubiquitous" symbol for autism (Grinker and Mandell 2015). As one autistic woman noted, "[the puzzle pieces] symbolize so much of what is wrong with popular autism discourse—representing autistic people as puzzling, mysterious, less-than-human entities who are 'short a few cognitive pieces,' who are utterly self-contained, disconnected, and need to 'fit in'" (Heilker and Yergeau 2011b). In a large study examining the social meanings attached to puzzle pieces, researchers discovered that "participants explicitly associated puzzle pieces, even generic puzzle pieces, with incompleteness, imperfection, and oddity", concluding that "if an organization's intention for using puzzle-piece imagery is to evoke negative associations, our results suggest the organization's use of puzzle-piece imagery is apt" (Gernsbacher et al. 2018).

Genetics and Autism

I would be remiss here not to note that many of these concerns follow along similar lines to those surrounding autism research and genetics. Concerns in the field of genetics in the autism community have largely been tied to fears about potential eugenics projects, whereby the ability to prenatally detect autism using non-invasive prenatal genetic testing would lead to a diminished number of autistic people being born, much as has been the case with non-invasive prenatal testing (NIPT) for Down Syndrome. Such fears include diminished social attention to the value of existing autistic lives, the loss of neurodevelopmental diversity in the general population, and decreased access to services as the populations falls (Hens 2015).

Where epigenetics raises particular concerns that differ from those of the genetic era lies largely with the above-discussed question of environmental factors and blame. While genetics differences are perceived broadly in liberal spaces as an "accident" of birth – indeed, one of the commonly used metaphors for a genetic profile viewed as particular advantageous or disadvantageous is "winning/losing the genetic lottery" – the implication of environment and behaviour into epigenetics draws on concepts discussed in chapter two concerning disability as a morally problematic state of being.

However, the question of potential heredity of traits implicated in the development of autism is one that is common to both genetics and epigenetics. A prime example of how neutral ideas about heredity are harnessed as part of more value-laden rhetoric can be found in the Autism Speaks problem "MSSNG". MSSNG constitutes a large-scale effort on the part of both Autism Speaks and Google to sequence the DNA of 10,000 families that include an autistic member, partly in search of familial links in autism. In families with autistic children, we have long known that there is a familial connection, and there is a higher likelihood of the parents exhibiting some autistic traits as well, even if that doesn't rise to the level of diagnosis (Ritvo et al. 1985). However, the autism community has raised concerns surrounding the framing of this research, as well as its end-goals, given the organization's commitment to seeking out data to assist in the development preventative and curative mechanisms. This pathologizing approach is embedded in the title of the research itself; in December 2014, Autism Speaks generated the online hashtag #mssng as a way of arguing that autism suppresses an individual's potential (Parsloe and Holton 2018). Recalling the New York Child Study Center's ransom notes advertising, they further implied that to be autistic was, in its own way, to *be* missing:

> Though Autism Speaks states that the title and missing letters "represent the missing information about autism that the research program seeks to deliver" (2017), they also reference a long tradition of seeing autistic people as changelings that are somehow less than the original person they were born to be, "empty fortresses" devoid of an interior (Rosenblatt 2018, Bettelheim 1967).

In the area of autism and heredity, then there is a concern that as the research shifts from viewing autism as environmental to autism as genetic and finally autism as epigenetic, it continues to carry forth the problematic narratives on which autism research has been built.

Epigenetics and Autism

Already a great deal of research is taking place examining the epigenetic mechanisms at play in autism. Given its location at the intersection of genetics and environment, epigenetics researchers have shown particular interest in studying the causal roots of autism. Past studies have shown there to be a high degree of hereditability in autism, and a large number of genetic studies have identified potential candidate genes. However, each of these genes seems to play only a small role, or to play a role in conjunction with other factors (Abdolmaleky, Zhou, and Thiagalingam 2015). At the same time, multiple epidemiological studies have uncovered associations between prenatal exposures, such as maternal stress or infections during pregnancy, and the development of autism (Cattane, Richetto, and Cattaneo 2018). Notably, researchers have concluded that the neurodevelopmental process is a time where the fetus is particularly susceptible to epigenetic modifications, particularly because during the prenatal period development is tied to patterns of gene expression. Because of this, researchers have concluded that certain environmental exposures could impact epigenetic modifications that raise the possibility of developing autism (Weber-Stadlbauer 2017). Studies examining differences between autistic and non-autistic brains post-mortem have similarly suggested that epigenetics may play a role in autism, based on the identification of differences in gene expression (Cattane, Richetto, and Cattaneo 2018).

As discussed in chapter one, many of the existing studies on epigenetics in general have been carried out so far primarily in animal models; this is no different for autism, where studies of the epigenetic impacts of prenatal exposures that have been linked to autistic behaviors in animals (Cattane, Richetto, and Cattaneo 2018). This research has been controversial, in part because of the challenge of identifying an "animal model" for autism, as diagnosis of autism is generally predicated on the absence or presence of very human social traits. Nevertheless, these mouse models have been used to study prenatal stressors and autistic behaviors. In other models, the mice have been genetically modified to possess known autism candidate genes, so that subsequent measures of epigenetic markers, biochemicals, and morphological differences can be studied, not only in individual animals, but across generations in order to understand the heredity patterns of epigenetic modifications (Cattane, Richetto, and Cattaneo 2018, Babenko, Kovalchuk, and Metz 2015, Weber-Stadlbauer 2017).

Studies have looked at how exposures from infection, emotional stress, and microbiome dysregulation may lead to epigenetic modifications in inflammatory and immune response pathways; this is tied, in part, to the questions raised above surrounding autistic individuals' tendency toward digestive and inflammatory conditions (Abdolmaleky, Zhou, and Thiagalingam 2015, Eshraghi et al. 2018, Weber-Stadlbauer 2017). Finally, research is beginning to explore therapeutic targets based on an epigenetic model of disease by the application of molecules in inflammatory pathways in attempts to reverse the effects of prenatal stressors (Moos et al. 2016). This promise of epigenetic treatments represents particular interest above-and-beyond the potential for genetic treatments, because of the possibility the epigenetic differences may be more easily reversed than genetic ones (Abdolmaleky, Zhou, and Thiagalingam 2015).

Throughout all of this research, the autistic community continues to raise concerns about the subjects and directions of data collection and use, as well as the framing of the results; this carries on from historical disagreements between autistic individuals and researchers (Furfaro 2019). The language of dysregulation, deficiencies, and impairment are prevalent across epigenetics autism papers (Berko et al. 2014, Zhu et al. 2014, Yasui et al. 2011, Momoi et al. 2010). In 2014, a study reported disappointment in the autism community in the funding priorities of UK autism research, which focused on biology, treatments, and causes rather than the research on services, supports, and knowledge about autism prioritised by the community (Pellicano 2014). Kapp et al. also found that autistic individuals are less interested in a cure for autism than are non-autistic individuals, and are more likely to view autism as something biological than environmental (Kapp et al. 2013). This fits in with the idea from the neurodiversity movement that autism is a natural variation and part of self; concerns about environmental influences are viewed as pathologizing and unnecessary, and epigenetic research focused on identifying disease mechanisms rooted in environmental causes is seen as harmful.

Reproduction

These harms from research that focuses on environmental causes are deeply tied to personal responsibility discourse in epigenetics research. As discussed above, one thing that epigenetics adds to our understanding of biomedicine is that it shows us on a molecular level how deeply interrelated our health outcomes are with our environments. Because of this, some bioethicists are concerned about how the discourse around epigenetics might create a perceived moral imperative to alter your own environment for health. Because many epigenetic modifications happen in utero, epigenetics ELSI scholars worry that this burden is going to disproportionately affect mothers. A number of scholars are already turning their focus to this responsibility discourse around mothers,

examining the problematic extrapolation from animal models to human maternal behaviours, and the lack of attention paid to the contribution of fathers to their offsprings' epigenomes.

This problem is particularly pronounced in autism research. Although mothers have been implicated in responsibility for their children's autism throughout the history of our understanding of the condition, we can see how these historical conceptions of autism, even ones that we know to be wrong, are becoming embedded in new ways through epigenetics research. The combination of a clear familiar component and the similarly clear import of environmental factors in autism means that epigeneticists are keen to discover the links between the two. Of particular worry for epigenetics ELSI researchers is the problem of how these links may be communicated in a way that causes harm; consider, for example, the widely-cited Frontline with Jenny McCarthy; when scientific research is twisted and allowed to go unchallenged, it impacts parental decision-making. And when that information, as in the case of the ransom notes controversy, is delivered with a tone of fear, this impact is heightened.

This is particularly important to remember in light of the hope that epigenetics research will lead to increased public awareness and public policy impacting epigenetic health-related behaviours. As such, it is imperative that researchers engaged in communication on epigenetics and maternal environments pay close attention to how their research is reported on, and to the lines between informing, persuading, and manipulating. Broadly, we consider someone to be engaging in persuasion when they are using reasoning, and manipulating when they bypass a person's reasoning skills, including through the employment of stereotypes or stigmatization. Public health campaigns, by their nature, tend to be geared at least toward persuading (MacKay 2017). Public health campaigns that employ manipulation tactics, however, impair the target's ability to make genuine informed decisions; by undermining the target's ability to reliably interpret information

and reliably assess themselves as a knower, manipulation tactics, particularly in areas that target emotionally fraught decision-making (such as major parenting choices) damage the agent's self-trust.

While the ultimate outcome of a campaign using persuasion and one using manipulation may very well be the same, only one will have coerced the agent into acting. This is an element that is missing from conversations about how "maternal capital", as in Wells' description of epigenetically disadvantaged mothers as metabolic ghettos, or when the mother as a person is conflated with the maternal body as an environment for the fetus (Wells 2012). In perceiving the maternal body as a mere vessel for epigenetic manipulation, we miss an opportunity to engage with the mother as a rational agent in her own right, instead jumping directly to perception of the maternal body as a malleable environment. Moreover, in engaging in the kind of rhetoric employed by the New York Child Study Center and Autism Speaks, any communication immediately moves from persuasion to manipulation.

It is worth noting, as well, that these communications can act in concert, and that even value-neutral scientific communication does not occur in a vacuum. Close attention needs to be paid to the social and cultural context in which decision-making takes place. This means recognizing that people may experience multiple kinds of vulnerabilities, or have had their autonomy undermined in other areas of their lives because of race, or class, or disability. It also means being conscious of how we approach epigenetic criticisms of environments or behaviour that carry cultural meaning. The statement that a certain behaviour or exposure may be implicated epigenetically in the development of autism is something that can be framed in a reasonably value-neutral way, but it will always be *heard* within the context of a larger society that devalues autistic people and their experiences.

While my focus is on the harms that this rhetoric ultimately causes for autistic individuals, it is important to note as well the harm that it causes for the ability to make clear and informed reproductive choices. Reproductive choice is most commonly framed as the right to decide when and under what circumstances to have a child. However, reproductive choice and reproductive autonomy more broadly engage an entire range of questions surrounding access to services and care, such as the decision to have a hospital birth or a home birth, or the decision regarding whether or not to undergo prenatal testing. These types of choices are in turn influenced by factors such as education, power differences in relationships with one's family and one's physician, and social and cultural context (Dodds 2000). In addition to a person's individual and specific life context, however, these choices are influenced by the rhetoric and ideology that surrounds the concept of "motherhood" (Stoljar 2000). Stereotypes generated by preconceived notions of femininity and motherhood, ranging from pressure to have children to the idea that the baby should come before anything else, including the mother's health, contribute to difficulty in separating the autonomy of the mother from the well-being of the fetus or child (Goering 2009). Reproductive decisions are framed as not only morally fraught, but as identity-forming for the mother; to be an authentically "good" mother is not only make the "right" reproductive choices, but to be sure of what those "right" choices are (Laufer-Ukeles 2011). In the face of increased pressure surrounding the potential for these choices to epigenetically alter the child, particularly in a way that is seen as generating a child of less "value", pathologizing rhetoric may deeply impact a gestational parent's autonomy to make reproductive decisions.

Epistemic Injustice

I have discussed above some of the negative rhetoric that surrounds autistic individuals. This impacts autistic lives in a number of ways, including by shifting focus from needed resources

to the search for a cure. However, the development and entrenchment of these harmful narratives, I argue, is also in and of itself a form of injustice. In contributing to inaccurate stories about autism, epigenetics research that does not do the work to unpack the social and cultural context in which it is taking place actively contributes to harm in autistic individuals' ability to know and name their own lived experiences.

In her work on epistemic injustice, Miranda Fricker addresses the idea that a person can be wronged specifically in their capacity as a knower. This first-order harm is intrinsic to the experience of having one's credibility questioned due to prejudice or being systemically excluded from resources that would enhance self-knowledge. While there are many pragmatic implications to the denial of credibility and knowledge – in the context of autistic individuals, for example, the lack of ability to access resources, misdiagnoses, and impaired quality of life – Fricker distinguishes epistemic injustice from the practical injustice, such as access to resources that occurs because of the disbelief (Fricker 2007, 45). In this conception of justice, being allowed to tell one's story and having that story heard and believed are goods unto themselves, as is access to information that increases one's self-knowledge. Epistemic injustice includes impairments in both testimonial credibility and availability of hermeneutical resources.

Testimonial injustices occur when someone's credibility is questioned because of prejudice surrounding their identity. Fricker defines the type of stereotype that contributes to testimonial injustice as:

> A widely held disparaging association between a social group and one or more attributes, where this association embodies a generalization that displays some (typically, epistemically culpable) resistance to counter-evidence owing to an ethically bad affective investment. (Fricker 2007, 36)

She argues that certain kinds of prejudices contain an intrinsic quality of disbelief, where the stereotype negatively colours the listener's judgement of the storyteller's credibility.

Hermeneutical injustice, by contrast, refers to a situation where there an individual has “some significant area of one's social experience obscured from collective understanding owing to a structural prejudice in the collective hermeneutical resource" (Fricker 2007, 100). This can include situations where a gap in “collective interpretative resources” unfairly disadvantages someone as they try to make sense of their social experiences”; they are again harmed in their capacity as a knower, but this time because they are unjustly kept from knowing themselves to the fullest possible capacity (Kalman, Lövgren, and Sauer 2016).

Although Fricker’s work does not engage directly with epistemic injustice and disability as it does gender, a number of scholars have picked up on these threads and elaborated on them in their own work. In particular, they have noted that individuals with intellectual, cognitive, or developmental disabilities are commonly perceived as being persons with limited credibility (Kalman, Lövgren, and Sauer 2016). The stigma attached to the social identity of intellectual and cognitive disabilities means that people may have their trustworthiness judged to be lacking based on identity alone, leading to testimonial injustice (Kalman, Lövgren, and Sauer 2016). Disabled people in general are often denied epistemic authority, both systemically and individually (Scully 2018b, 109).

In the healthcare context, both Fricker and others have noted that a “prejudicial dysfunction in testimonial practice” (Fricker 2007, 164) may lead to biomedical experts having their credibility privileged over that of their patients *pro forma*, and for disabled individuals to have their self-knowldege discounted. Ho suggests that this epistemic hierarchy may be alleviated through the employment of “epistemic humility” on the part of medical professionals (Ho 2011, 117). Epistemic humility “recognizes the boundary of their own expert domain and the potential contribution of patients with impairments in knowledge creation” (Ho 2011, 103). As Scully notes,

however, although well-intentioned individual healthcare practitioners may work to become more "virtuous knowers", paying attention to the power imbalances that shape their belief in their disabled patients' narratives, this may make very little difference overall in the individual's epistemic experience without broader changes to "policies, organizational practices, and epistemological structures" (Scully 2018a). As a result, disabled individuals are "potentially exposed to unique forms of lifelong epistemic exclusion because the highly specialized epistemological structures of health and social care expertise often dominate their everyday lives" (Scully 2018a).

Although epistemic injustice in the context of autistic individuals was not a creation of epigenetics, it has certainly been exacerbated in this field, particularly in the area of hermeneutical injustice. The history of autism research has been one of push and pull between questions of nature and nurture, but always with an underlying prejudicial stance that considers the existence of the autistic individual to be not just the *result of* one of these two, but the *fault.* From refrigerator mothers creating autistic children through cold home environments to the MSSNG project and its claims of children "stolen" from their families by genetics, the narrative has remained one that views autism as a flaw to be avoided or corrected. In doing so, it has also dampened or silenced the narratives from autistic individuals themselves, about their rich and complex lives, and the potential joys found in autistic traits. Epigenetics researchers did not create this injustice, but as they move forward in communicating about the joint efforts of nature and nurture that go into the development of autistic individuals, they are uniquely positioned to help rectify it.

Conclusions

In this final chapter, I have elaborated on the history of pathologization, shame and blame involved in both autism research and media reporting on autism, beginning with the causal

assumptions made by early autism researchers. My intention is for this chapter has been to allow it to function as a proof of concept for chapters one and two, to provide a concrete cautionary example of the impact of pathologization on disabled individuals, and the way that epigenetics research is helping to further and entrench this pathologization.

Initial conceptions of maternal responsibility for autism in the early days set the stage for more modern discourse of shame and responsibility. Epigenetics research is in some ways bringing us full circle, from that early rhetoric around the home environment and emerging understandings of genetics and heredity to the understanding of how these function in tandem. As we grow into a clearer understanding of the epigenetic implications of autism, we also run the risk of taking the worst of past stigmatization and combining them into a narrative that appears to be new, on the surface, but nevertheless fails to break free from these historical concepts. In particular, the idea of autism as a function of maternal negligence, while long disproven, has proven to be a trenchant example of a proto-idea that has followed us into twenty-first century science.

As discussed in chapter two, lenses from disability theory can help us to understand exactly why the pathologization of a condition, as well as the search for its "causes", can be problematic. Tracing the anxiety surrounding maternal responsibility for disability back to early examples that situated the causes of autism in "bad mothering" serve to show how intransigent the discourse on responsibility for inherited disability will remain so long as the societal fear and disparagement of disabled bodies is not questioned in parallel. Autistic individuals experience real harm from the application of the normative values that have accompanied the "deficit" model of cognitive well-being. Incautious communication about epigenetic autism research risks exacerbating existing harms to autistic individuals and their families that have been linked to the stigmatization of autism, including high rates of depression, anxiety, and suicide, as well as caregiver abuse

spanning from potentially fatal curative attempts and infanticide (Ghaziuddin, Ghaziuddin, and Greden 2002, Richa et al. 2014, Edelson 2010, Schopler 1994).

I wrote this chapter starting from a framework that presupposes the value of a neurodiverse population, and have focused on this history of autism research less for its implications, and more for the narrative it presents. Continuing with this theme of stories told and untold, I end on the note of epistemic injustice. This is not necessarily the most pressing of all of the harms that emerge from pathologizing autism, but it is maybe the most personal; epistemic injustice in the form of masculinized narratives that gatekeep girls and genderdivergent individuals from diagnosis kept me from finding my own place on the spectrum until well into law school, when a great deal of damage to my mental health and sense of self had already been done. I am well now, finally, and trace the history of my own harms not to my neurodevelopmental differences, whatever their origins may be, but to having been time and again given an incomplete and inaccurate story of my self. This is an experience I share with many on the spectrum. Ultimately, I have found that many of the brilliant autistic minds that I surround myself with now are far less interested in how we came to be than in who we have become, and in how to build a better future for existing our autistic community and those who will follow.

Conclusions

It remains to be seen whether epigenetics will continue to emerge as a field unto itself, or be subsumed as a sub-category of genetics or molecular biology. However, what is clear is that epigenetics provides us with an opportunity to examine how scientific narratives and their associated moral categories emerge. Moreover, it provides us with a model of how disability theory can be incorporated into an emerging science from early in its development. Although epigenetics is not conceptually new, both scientific focus and public interest have intensified in recent years (Dupras, Saulnier, and Joly 2019). Now is the time to address issues in the field, and to course-correct where necessary.

By questioning the standards by which a society includes or excludes certain bodies from the definitions of "healthy" or "normal", disability theory can provide insight into the language of epigenetic "harm" that undergirds the discourse on epigenetic responsibility. As discussed in chapter two, biomedicine is often not just involved in the assignment of "health" values to different conditions and different bodies, but to moral values as well. With each shift in our understanding of human biology come new attempts to assign normative value to people and their bodies. The genomics era shifted the focus on health and wellness to the molecular level, bringing with it the language of "good genes" and "bad genes" as shorthand for desirable and undesirable bodies. Now, in the post-genomic/epigenetics era, researchers are moving toward a framework that rejects rigid conceptions of the body in favour of understanding the body as mutable and permeable, intrinsically tied to its environment. It stands to reason that, unless we are careful, those environments – including the humans who reside in and interact with them – risk becoming stigmatized as well.

Epigenetics is often described using metaphors of light switches (or, for more nuance, dimmer switches). The genome determines the architecture, while epigenetics determines which rooms have their lights turned off or on. I tend more toward literary metaphors, not the least because the first time the genome was explained to me in a way that clicked, it involved the mapping of the human body as the discovery of a text, a text where we have only just begun to piece together words into sentences, like children learning to read. Epigenetic processes, in squeezing the text to make certain bits unreadable, function as an additional challenge to our reading of genes, but also present hope for their modification; unlike the stubbornly static genome, the epigenome shifts from outside influence. As any writer can tell you, though, editing is a tricky process. You put all your thoughts on paper to start, and then try to separate out only those words that benefit your story.

The story of epigenetics has yet to become fully clear, but it is being framed as something exciting and new. Pickersgill argues that epigenetics is being "constructed as an area of biomedical novelty", leading in part to enthusiastic but ambivalent promises being made on the part of scientists (Pickersgill et al. 2013):

> Waddington is a frequent reference point in review articles and editorials on epigenetics, serving as a means of 'colonising the past' that establishes the legitimacy of the field and is suggestive of conceptual and empirical advancement in ways that invite anticipation of developments not yet made. The act of pointing to a lineage implies that "the" origin of epigenetic research is unfamiliar to many scientists; hence it must be a little quirky, a little beyond the mainstream of biomedicine. (Pickersgill et al. 2013)

The hype and hope attributed to epigenetics is appealing, but for every researcher who claims that epigenetics changes everything, another comes along to argue that it is merely a crystallization of things that we already knew about the genome and the environment. Or, as Jonathan Huang and Nicholas King argued in the controversial 2018 paper of the same name: "Epigenetics Changes Nothing" (Huang and King 2018).

What, then, do we make of epigenetics? Is it, as some bioethicists fear, merely a further act of bio-reductionism, dragging our bodies and their surroundings down to the unknowable (except by specific, siloed expertise) molecular level? Or is it the ultimate answer to the question of nature and nurture, once and for all showing that neither the biological nor the social can be examined in a vacuum? No matter the answer to those questions, we can convincingly argue that epigenetics provides us with new perspectives, new modes of understanding, and new implications for human health. It will remain to be seen whether the field recognizes the cautionary examples of the past to break out of the pathologizing narratives of its predecessors, or carries them on to a new era of science.

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